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THE OTOLOGIC MANAGEMENT OF PROGRESSIVE DEAFNESS.*

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Your Secretary, in assigning to us this title, requested that this paper be confined to the consideration of the otologic management of progressive deafness, excluding otosclerosis, deaf-mutism, etc. We define progressive deafness as that form of deafness consisting usually of both conduction and perception loss in variable amounts, characterized, as its name implies, by a tendency toward further steady or intermittent loss of hearing, of very variable etiology and with or without a history of previous suppuration. In other words, if we leave out of consideration, as we must, otosclerosis, the term applies to almost all other kinds of deafness of more or less insidious origin and course that the otologist is called upon to "manage."

We well appreciate that many prominent otologists believe that most cases of deafness that show true and marked progression are of otosclerotic origin, although otosclerosis itself does not always progress uniformly or beyond a given point and, indeed, may be present without demonstrable loss of

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hearing. We also know that it is accepted as a fact by many that deafness from other causes except, perhaps, that accompanying senility, does not progress to any great extent, especially those types of so-called tubotympanic deafness originating in childhood. Nevertheless, many cases of deafness, or of hardness of hearing, which come to the otologist for help, cannot be definitely put in the class of otosclerotics, and for them we must do as much as lies in our power. If untreated, or improperly treated, these are likely to become progressively worse. Even under the very best treatment, this condition will often advance to a stage where all hope of improvement is lost. Conversely, good treatment has often not only arrested a rapid decline in hearing acuity, but even sharpened up audition to an appreciable degree.

The seriousness of the problem before us need hardly be stressed before the members of this distinguished Academy, for it has often been stated that next to blindness, deafness is the greatest misfortune that can befall any human being.

Dickie,[1] summarizing the bibliographic material available in the field of otolaryngology, states that there are about three million deafened school children in the United States.

Randall,[2] some years ago, estimated economic usefulness to be reduced as follows: 25 per cent where the conversational voice is not heard beyond one meter; 22 per cent when heard between one and five meters; 11 per cent when heard between five and ten meters, and 5 per cent when heard between ten and 20 meters, and that "it must not be forgotten that many cases of deafness even under the best treatment are apt to grow progressively worse." The three fundamental principles in the otologic management of progressive deafness may be set down as follows: *1.* The institution of measures calculated to prevent deafness; *2.* Measures that will aid in the conservation of hearing; *3.* Careful testing of the functional capacity of the acoustic apparatus from time to time.

1. Preventive Measures: Do we admit that hereditary deafness is always otosclerosis? Congenital deaf-mutism is often hereditary, and according to J. K. Love, follows the Mendelian law. Certainly other cases of progressive deafness, appearing later in life, often give surprising family histories of deafness. Must these all be classified as otosclerotics on history alone, if other diagnostic signs are not apparent? If we

agree that a tendency towards any other type of deafness may be transmitted to offspring, then a very obvious precaution would be for the otologist to advise against the marriage of those persons with bad family histories of deafness, especially if either, or both, of the contracting parties is deaf. Unfortunately, we are not often asked for advice on this matter, or if given, the advice is frequently ignored.

Every otologic case should be thoroughly studied. A careful history, a general examination and a thorough local examination may lay the foundation for the institution of proper therapy. Dean,[3] with his characteristic thoroughness, says that it takes time to get a proper conception of the factors which bear upon deafness, hence he finds that it requires a minimum of 20 minutes to get an otological history. The family history of deafness should be carefully inquired into, for where an hereditary factor exists, the acoustic apparatus of such an individual already is a "locus minoris resistantiae," more vulnerable to deleterious influences than a normal acoustic mechanism would be.

Because of the limited time allotted to this paper, we may but briefly summarize some of these influences. Syphilis, hereditary or acquired; acute contagious diseases, exanthemata, all upper respiratory infections, poor health habits, metabolic faults, nutritional imbalance as pointed out by Stucky,[4] endocrine imbalances reported by Drury,[5] the intermarriage of persons with a family history of deafness, the harmful effect of noise upon the delicate organ of Corti as emphasized by Wells,[6] harmful drugs, such as arsenicals, quinine and salicylates (on the perceptive apparatus); swimming, diving, improper nose blowing, middle ear suppurations, infection of the nasal accessory sinuses, infections in remote parts of the body, such as those in the gastrointestinal or genitourinary tract, dental infections; these are some of the influences.

To prevent irreparable damage to the delicate auditory nerve mechanism, it is necessary to minimize all these conditions as far as possible by further researches into the underlying causes of progressive deafness that still remain obscure, by closer co-operation between the medical profession, public health authorities, industrial managers, school authorities, social service workers and the laity, by educational propa-

ganda awakening the mind of the masses to the importance of good hearing and by adequate legislation to secure the desired results. A more detailed description of these points appeared in a paper read by one of the authors[7] in 1931. It is a *sine qua non* that meticulous and skillful treatment of the nose and throat during and after any contagious or respiratory disease will go far towards preventing aural complications with consequent deafness. These methods will be described under the caption of Conservation of Hearing.

Years ago, Emerson[8] taught that a primary focus of infection exists for every case of progressive deafness. He believed that this primary focus may be subject to repeated acute exacerbations or remain quiescent, running a chronic course, but elaborating toxins that were slowly absorbed, producing auditory damage. In other words, the entire picture was to be looked upon as one of toxemia, septicemia or pyemia, running an acute or chronic course. The focal processes were often multiple, and were to be found in the teeth, tonsils and sinuses, in all the lymphoid structures of the epipharynx, oropharynx, laryngopharynx, as well as in and around the lateral pharyngeal folds and Rossenmuller's fossa. One must not overlook the intestinal tract, the appendix, the gall bladder or the colon. Later Emerson[9, 10] stated that, essentially, the perceptive apparatus is the mechanism involved in progressive deafness and the focus might be even a closed follicle in a chronically infected tonsil. Furthermore, he taught that pathology in the tube and middle ear had little relation to nerve dysfunction. Emerson[8] advised the removal of every vestige of infection, particularly emphasizing the infective processes in the nasopharynx, lymphoid tissue and bands between the Eustachian tube orifice and the pharynx, and degenerative tissue in Rossenmuller's fossa, as well as thorough removal of the faucial tonsils.

Where defective sinus drainage exists, free, spontaneous, permanent drainage (to quote Herbert Tilley) should be instituted at once. This is accomplished by reconstruction of the nasal chambers, correcting badly deviated septa, removing redundant tissue from the turbinates, reducing posterior hypertrophies and opening all infected cells. The nasal accessory sinuses should be studied clinically as well as by roentgen ray, and the latter study augmented by use of radiopaque oil

injected directly into the maxillary sinuses and instilled in the posterior ethmoids and sphenoids by the displacement method of Proetz. By these measures, a latent sinusitis may be discovered, the removal of which might well prevent a certain amount of deafness. Emerson[11] has again emphasized the importance of the maxillary sinus as a focus of infection which coincides with our observations. Mosher[12] feels that although we can by routine examination estimate the gross amount of infection in a given antrum, we can by no definite means evaluate the ability of any given mucous membrane to produce toxins. Furthermore, it is not the amount of toxins but their virulence which determines whether the effect caused by them is local or general. He does not believe that pus and polypi in the antrum cause very much trouble, but are as a rule self-limited affairs. Lastly he states that where a mucous membrane has reached the atrophic stage it usually becomes innocuous, incapable thereafter of acting as a focus of infection. Emerson[11] takes the stand that even an atrophic mucous membrane, as well as its periosteum, may act as a focus of infection.

Otology owes much to Dean[13] for his excellent work on the management of Nasal Sinus Disease in Small Children even if we only consider it in the light of prophylaxis for later developing deafness.

In connection with the factor of foci of infection, French[14] strongly emphasized the lingual tonsils, the infratonsillar nodules and the lymphoid apron. He and his associates performed an "expanded tonsillectomy" to remove these infected lymphoid elements.

Sturm[15] has been impressed with the fact that chronic progressive deafness begins in childhood. He stated that he had rarely seen a perfectly normal drum in a child with adenoids. He divided adenoid deafness into three stages: occlusion, transudation and cicatrization. In a study of 200 patients suffering from deafness, he found 32 per cent owing their incurable disability to nasopharyngeal disease in childhood.

Thus far in our essay we have searched for the various etiologic factors concerned in the production of progressive deafness and we have reason to believe that we have found, chiefly, multiple foci of infection situated in the nose, throat, mouth,

and distant foci, besides extraneous environmental conditions to account for the deafness. It remains for the otologist to search carefully, systematically and thoroughly for every possible cause with a view to removing that cause. By removing the cause we may prevent the effect.

So much for the preventive measures.

2. Conservation of Hearing: All measures designed for the prevention of deafness constitute the first and basic principle of conservation of hearing. When the causative factor has been removed, the perceptive apparatus begins to show improvement provided that it has not suffered irreparable damage. The question of local measures must now be considered. Here the old axiom: a thousand men, a thousand minds, holds true. It is interesting to note the diverse views expressed by our eminent colleagues. Shambaugh[16, 17] teaches that progressive deafness in adults has, with very few exceptions, nothing to do with tubotympanic alterations which constitute the type of ear trouble which might logically be blamed upon nose and throat conditions. He contends that in adults, progressive deafness is the result of alterations which take place primarily in the bony capsule of the labyrinth. Therefore, he is averse to any treatment directed to the nose, nasopharynx and Eustachian tube as well as correction of septal irregularities, spurs, ridges, hypertrophies, hyperplasias, etc. Freund and Hays[18] warn against excessive politzerization, claiming that it may actually do harm. Emerson likewise is of the opinion that inflation is of no value and that if practiced as a routine, may cause the diseased condition to progress further.

Bruhl and Politzer[19] state that the mechanical effect of a current of air entering the tube is as follows: The lumen of the tube is dilated, whereupon any secretion that may be present in the tympanum is enabled to escape; that the lateral pressure produced within the tube during politzerization forces the blood out of the hyperemic vessels and thus diminishes inflammation and regulates the circulation in conditions of stasis. Furthermore, when the ear drum moves outward the chain of ossicles also moves outward and abnormal tension is removed while inflammatory adhesions are broken up, blood and lymph vessels of the tympanic mucous membrane regain their normal pressure, absorption of exudate is facilitated and the intralabyrinthine pressure is regulated. It

is apparent that a mild stimulation takes place during inflation.

It has been our observation that where removal of infected foci is combined with judicious inflation or tube treatment, we have obtained the best results in treating progressive deafness. We wish to sound a warning against indiscriminate removal of diseased foci as well as haphazard unconsidered surgery. We believe, however, that elimination of infection from the body, as far as possible, is conducive to general bodily health and thus indirectly, if not directly, to aural health, and so is often justified.

The technique of the inflations which has given us the best results is as follows: mild shrinking of the nasal mucous membrane with a spray followed by a cotton tipped applicator dipped in a mild solution of ephedrine, epinephrine or cocaine and antipyrine, and applied to the pharyngeal end of the Eustachian tube. The silver catheter is carefully inserted so that the tip fits accurately into the tube mouth. A small electric heater is attached to the electric circuit and a cut-off from the air pressure line fits into a small aperture in the proximal end of the electric heater so that the cut-off can be regulated with the thumb. The distal end of the heater is provided with a chamber containing a small quantity of iodine crystals, camphor, menthol and talc (Vansant's formula). Thus by applying pressure on the cut-off, air is driven through the electric heater so that vaporization takes place over the iodine crystals. The tip of the electric heater fits snugly into the mouth of the Eustachian catheter. Thus medicated vapor is provided, which passes through the tube and into the middle ear. Pressure on the cut-off is performed rhythmically, and in this manner the hot air is driven through in small interrupted puffs and a minimum pressure is always used. This is a good guard against overinflation. The diagnostic tube likewise is always employed. By feeding the hot air in interrupted puffs to the tympanum a vibratory effect likewise is produced. After inflation, the catheter is removed and a mild astringent, such as colloidal silver or silver nitrate, 2 per cent, is applied to the pharyngeal tube mouth. Treatments are given semi-weekly or oftener for a number of weeks. All of our cases for the past number of years have also received some vibratory massage to the drums by means of an electrically driven oscillator.

Where constriction or marked stenosis of the tube exists, it is necessary to use some means of dilating the tube. This is accomplished by means of Eustachian tube applicators and bougies. After careful shrinking of the tube mouth, an applicator of the Yankauer type is wound with a small amount of cotton for about two inches from the end. This is saturated with 10 per cent cocaine solution or 3 per cent ephedrine, passed through the catheter to and beyond the tubal isthmus and kept there for about a half hour. It can then be replaced by another applicator dipped in silver solution, or a slightly larger size bougie can be inserted and allowed to remain *in situ* for some time. The heated bougie demonstrated at this meeting by Simon may well be an aid. At no time must much force be applied in any of these treatments. After the Yankauer or bougie treatment, no inflation should be done, and the patient cautioned against forcible nose blowing.

Where there exists an atrophic type of middle ear deafness and where the Eustachian tube is patulous and the drumhead relaxed, no inflations should ever be done. The patient should likewise be cautioned against forceful nose blowing. He should be instructed to blow the nose gently with either one or both nostrils open. The drum head may respond temporarily to various agents used to tighten it up, *e. g.*, collodion applications, cantharides in collodion, or myringotomy, though all of these may fail and a perforation result.

High frequency currents may be tried. Hays has a glass electrode shaped like a Eustachian catheter. This is wrapped in adhesive to the tip to obviate injury if the glass breaks. This electrode is inserted exactly with the same technique as for the catheter and the current applied for about five to ten minutes. It is said to decongest the tubal mucosa.

Diathermy applied to the middle ear through the external canal by electrodes may help in absorbing exudates and adhesions. X-ray treatment for the restoration of hearing is of doubtful value. Its beneficial action, if any, would seem to be in reducing lymphoid elements about the pharyngeal orifice of the Eustachian tube. Mackenzie[20] has also tried diathermy in chronic cases with such satisfactory results in diminishing deafness and tinnitus that he has expressed the feeling that it is worth a further trial.

Where aural suppuration is present and hearing is gradually falling off, every effort should be made to control the suppuration and get the ear dry. Attention to the nose and nasopharynx, careful, conservative treatment of the ear with time proven methods will in most cases improve the condition as well as the hearing.

No commercial drums should be used. The aurist can make a small disc from glazed paper, slightly larger than the perforation and apply it to the drum. This will often improve the hearing. A very small piece of cotton, shaped as a ball, cylinder or cone, can be applied through the perforation to the wall of the middle ear in the region of the oval window. For ease of insertion, the cone can be dipped in collodion from the point almost to the base, thus rendering its introduction, as well as extraction, easier for the aurist or patient. When moistened, as with glycerine, this simple device at times acts as an efficient hearing aid and is conducive to the comfort and morale of the patient.

It is of the utmost importance to conserve the hearing in infancy and childhood so that the integrity of the acoustic apparatus may be retained to an efficient degree throughout life. Since we believe many cases of progressive deafness in adults are an aftermath of inflammatory states of the Eustachian tube and middle ear in infancy and childhood, the closest co-operation between pediatrician and aurist is necessary in all cases of contagious diseases, acute exanthemata and respiratory diseases. The children should be frequently examined. During acute attacks of any upper respiratory infections, careful attention should be directed to the nose and nasopharynx, mild shrinking solutions, warm alkaline solutions and any form of colloidal silver may be instilled with a medicine dropper, or a mild shrinking solution followed by very gentle glass suction, followed by a bland oil, will be found efficacious.

Recurrent attacks of upper respiratory infections can be curtailed by a surgical cleanup of the nasopharynx, removal of the pharyngeal tonsil as well as the degenerated lymphoid tissue in and around Rosenmuller's fossa. After any upper respiratory infection has well subsided, it is important to ventilate the middle ear cavity and invigorate the drumhead by gentle inflation with either a Politzer bag or a nebulizer for

a number of treatments, as well as to massage the drum and pinna.

The resistance of every individual, whether actually or only potentially deafened, should be raised to the highest level. This will, to a certain degree, prevent colds and sinus infections. Good fresh air, sunlight, good food, proper clothing, proper breathing habits (full deflation and inflation of the lungs) and fractional doses of good stock of autogenous vaccine will help to ward off respiratory infections and conserve hearing. The avoidance of all nose washes (Dobell's, Seiler's, soda), the interdiction of hard nose blowing, habituation to cleaning the nose by suction down the nasopharynx or by negative pressure, and the gentle stimulation of the neurovascular system by regular suitable setting up exercises as recommended by E. R. Lewis[21] likewise will improve the health and resistance, both general and local, of the patient. Nasal hygiene, as recommended by Culbert[22] years ago, is a valuable adjunct. It consists of exercises whereby the expired air is driven out with a little force, *i. e.*, forceful outward breathing is performed. This rational procedure drains the sinuses better and improves the function of the nose. It has been well commented upon by Goldsmith[23] and Beck.[24]

Where the deafness has progressed to such an advanced degree that it fails to respond to any treatment, various sound amplifying devices should be resorted to. These patients should be encouraged to go to theatres, to listen to music, to attend to various activities. This exercises any hearing that may remain and disuse atrophy is thus retarded. It is important that such individuals attend meetings of the various Leagues for the Hard of Hearing. They should also be taught speech and lip-reading. From time to time they should receive careful tube treatments to relieve the tinnitus and the sensation of "fulness in the head." Every effort should be made to improve their morale and the psychologic factor of cheering the patient and putting new heart and spirit into the incurably deaf should never be forgotten by the aurist, as Berry[25] has so recently and forcibly impressed upon us.

3. Functional Capacity of the Acoustic Apparatus: Newhart[26] states that the prevention of deafness on any comprehensive scale must include the periodic testing of the hearing of all public school children by modern methods and that

periodic health examinations of persons of all ages should include hearing tests adequate to disclose any significant loss of hearing acuity. It has been our feeling that when careful tests are instituted from time to time, the aurist is in a better position to judge the integrity of the acoustic apparatus. This is very important in the otologic management of progressive deafness.

In testing the functional capacity, it is well to utilize all the standard methods, *i. e.*, conversational and whispered voice tests, watch, low, middle and high fork, Weber, Schwabach, Rinné, Galton Whistle and the Audiometer. The new audiogram blank of Newhart[27] is well adapted to this end. For a more detailed account of the Functional Testing of Hearing the reader is referred to Sonnenschein's[28] excellent description.

Of course, it is obvious that, as many of these cases, if not most of them, are insidious in their onset and progress, the earlier the condition is discovered the more chance there is of taking some effective measures for their relief. In the interests of efficiency, it will not do to wait until the disease has progressed to the point where the patient is himself aware that hearing is diminishing. This may be long after irreparable damage has taken place and indeed, to make matters worse, many individuals, when impaired hearing is first noticed, will, through inertia, fear, lack of time or opportunity or for many other reasons, postpone their visit to the aurist, often hoping that the condition is but transitory. If there be any virtue in general periodic health examinations, and we believe that there is, then a regular periodic aural health examination, even after the school age, would seem to be justified.

Conclusions: The otologic management of progressive deafness is best conducted by searching for all etiological factors capable of causing the deafness and removing those factors as far as possible, as well as keeping the nose, nasopharynx and tympanum in the best physiological condition. When deafness supervenes, suitable cleanup and local measures should be instituted and all hygienic measures utilized. Where the deafness is incurable, suitable hearing aids should be recommended, the study of lip or speech reading urged and the attendance upon Hard of Hearing Clubs advised. The aurist

should never relax in his endeavors to keep up the courage, morale and spirit of the deaf one.

From time to time thorough functional testing of the acoustic apparatus should be performed, utilizing all the standard tests, in order to know the progress of the disease.

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INFECTION OF THE PETROUS APEX.*

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Petrous apex infection refers to infection of the petrous pyramid, complicating an acute suppurative otitis, with or without a surgical mastoiditis.

Only recently has much been written on this subject.

In 1930 and 1931, much was added to our knowledge of this condition by the work of Friesner and Druss, Kopetzky and Almour, Eagleton and others, Friesner and Druss reporting 17 cases; Kopetzky and Almour, nine; Eagleton, six; C. H. Smith, four; Bowers, two, and Profant three of his own and 18 from the literature since 1924.[1]

It can safely be stated that at least one of the triad of the Syndrome of Gradenigo, most often two and often all three, present during the progress of apex infection, intervening between the period when the infection is apparently superficial and the time when grave intracranial complications appear evident. That is, we may say that many cases presenting Gradenigo's symptom complex are infections of the petrous apex, although all cases of petrous apex infection do not present Gradenigo's Syndrome.

In 95 case reports of paralysis of the sixth cranial studied by Perkins,[2] the cause in 33 cases was given as follows: sinus thrombosis in two, meningitis in three, labyrinthitis in four, abscess in the posterior fossa in nine, abscess in the middle fossae in two and petrositis in 13.

When one considers, however, that all of these causes, except perhaps labyrinthitis, might have been preceded by an apex infection, this brings a good deal of evidence to bear that a very large number of cases presenting Gradenigo's Syndrome are infections of the petrous pyramid.

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Eagleton states that this type of infection can produce a local, microscopic, extradural abscess in the floor of the middle fossa, the result of a superficial erosion of the superior surface of the petrosa.

Most authorities believe that invasion of the petrous apex is the result of direct extension of the infectious process, associated with extensive pneumatization.

Kopetzky states that in all of his cases, with involvement of the fifth and sixth nerves, there was either operative or X-ray proof of extensive pneumatization. Eagleton[3] is of the opinion that the infection takes place by way of blood stream and looks upon the pathology as an osteomyelitis, since the petrous apex contains marrow similar to that of a long bone.

The simplest classification of routes of invasion is that of Profant, who recognizes two routes, the antrotympanic and the hypotympanic route.

The clinical picture of petrositis may present during the progress of a middle ear suppuration, which may not be complicated by a mastoid suppuration, or it may occur during the postoperative period.

Kopetzky[4] divides symptoms into four periods: period of eye pain and aural discharge; period of low grade sepsis; period of quiescence, and terminal stage.

Pain is, of course, on the side of the lesion and referred to the area above the eye and within the contents of the orbit itself. This pain is often more intense at night. As a rule, discharge from the ear is profuse and quite constant, although there seems to be, occasionally, a relationship between the intensity of pain and diminution in quantity of discharge.

Phillips[5] states that dull, aching pain felt on the side of a head, some time after operative interference on the mastoid, signifies a lateral sinus thrombosis in contradiction to the pain referred to the ophthalmic branch of the facial in apex infection.

Many uncomplicated simple mastoid patients present low grade temperatures for some time, but if with profuse dis-

charge, which does not seem influenced by the surgical procedure already executed and associated with ophthalmic pain, apex infection must be given serious consideration.

The period of quiescence, if present at all, varying from five to 19 days, is the most important period from the standpoint of the patient. During it one must determine whether the infection of the petrous apex will find a satisfactory exit, or it is that period preceding serious intracranial extension.

The terminal stage is the one of rapidly progressive meningitis or brain abscess.

The infection may find a satisfactory exit, by way of sinuses draining into the antrotympanic cavity, or around the Eustachian tube in the middle ear, or through the same avenues through which it traveled inward, if by way of direct extension. Infection may also break through the cortex, on the superior or posterior surface of the petrous, causing an extradural abscess of the middle or posterior fossa.

Most often the infection is stamped out by the patient's natural resistance, to use Eagleton's words, "the bone spontaneously controls the infection, because of its highly reactive character."

This statement explains the reason for the high percentage of recoveries in cases presenting Gradenigo's Syndrome, the mortality of which is estimated to be from 8 to 16 per cent.

The fifth nerve pain is explained as the result of a protective inflammatory reaction, within the meninges with edema of the region of the superior surface of the petrous apex and the petrosphenoidal articulation, causing pressure very often early, on the ophthalmic branch of the facial, which is not only the longest of the three branches given off the Gasserian ganglion, which lies in a depression provided for it on the superior surface of the pyramid, but is the only one bound down by dura, making it, for these mechanical reasons, most likely to respond early to any increase in pressure.

The sixth nerve with the superior petrosal sinus, traverses Dorello's canal, which is formed by the petrosphenoidal ligament, as it passes from the apex of the petrous to the posterior clinoid process. It is in this region that the sixth nerve is

caught in this inflammatory reaction, explaining the failure of function of the external rectus.

There is nothing in the bacteriology of this complication of the middle ear disease in the blood, spinal fluid or eye grounds common or characteristic.

Sixth nerve palsy is not a constant symptom, nor necessary to establish a diagnosis.

In the Friesner-Druss[6] series of 17 cases, there were 16 sixth nerve paralysis and two of the seventh, while in the Kopetzky-Almour series of nine cases, there was but one sixth nerve palsy and three of the seventh.

Eagleton's series of six showed three sixth nerve paralysis, and all four of Smith's. Bowers' two cases showed no sixth nerve paralysis, but one paralysis of the seventh.

Of this group of 38 cases, there were 24 of sixth nerve paralysis.

The outcome of this type of infection seems to depend upon many factors, as we find complete recoveries following only the simplest surgical procedures. If you are persuaded that Gradenigo's Syndrome is caused by a combination of an infected congestive lesion of the apex cells and a protective reaction in the meninges, your belief is that 84 to 92 per cent of all cases of petrositis recover without special surgery directed toward the petrous apex.

Of the Friesner-Druss[7] series, there were seven deaths and ten recoveries, treated as follows: 13 simple and one radical mastoid operations; one myringotomy; two had only spontaneous ruptures. Of the patients treated by simple mastoid, five died; the radical and the one treated only by myringotomy died.

Of the Kopetzky-Almour series, there were five deaths and four recoveries. Six of these cases were treated by a new operation developed by these men, with 50 per cent mortality.

Of the Eagleton series of six treated by him with his newest operation, which he calls "unlocking the petrous pyramid," there were three recoveries.

Two of Smith's[8] cases treated by simple mastoid operations recovered; the third, by a radical, recovered; the fourth, a

radical, died suddenly, presumably from an embolus. In these last two cases, extradural abscesses were evacuated after cerebral exposure and inserting a dressing forceps downward, forward and inward, hugging the anterior surface of the petrous portion of temporal bone.

Both of Bower's[9] cases recovered with radical operations.

Including two cases reported in March by Lillie and Williams[10] which recovered following secondary simple mastoid operations, the percentage of recoveries in this group, treated by surgical methods already in our hands, were higher than those treated by surgical measures more recently developed. There can be no question, however, that if these simpler measures fail there is need for a method of surgical approach into the pyramid which will be within the reach of all of us.

Many of these radical operations were conversion operations from primary to secondary operations. In most cases definite sinuses were found, leading into the petrosus, which if not exposed by the simple mastoid operation, were by the radical.

Death, except in the one case of embolus, was either from meningitis or brain abscess, a few showing signs of septicemia.

At the January meeting of the Section of Otology of the New York Academy of Medicine, Kopetzky and Almour presented a new method of opening the petrous pyramid. It consists of a trephine opening by a small electrically driven burr driven into the petrous pyramid through the superior anterior angle of the inner wall, in front of the cochlea and the facial nerve.

In the March number of the *Archives of Otolaryngology*, Eagleton describes his method of unlocking the petrous pyramid. This calls for an extensive removal of the anterior buttress, consisting of the zygomatic root, forward to the articulation of the mandible, together with the tegmen of the tympanum and the tegmen antri and the tegmen cellulae, enabling a free elevation of the dura covering the floor of the middle fossae, and inspection of the anterior surface of the petrous pyramid. He also removes of the posterior buttress

sufficient bone from the mastoid and the superior petrosal regions to permit reflection of the sigmoid sinus slightly backward, giving an exposure to the bone of the posterior surface of the petrous pyramid, opening the apex with a hook. Both Kopetzky and Eagleton agree that only drainage is necessary.

As I think back over my cases of suppurative middle ear disease that have been complicated by meningitis, brain abscess and cerebral sinus thrombophlebitis, I have often been at a loss to explain the reason for their development.

I believe that I have had in my work at least four infections of the petrous apex, two of which recovered following secondary simple mastoid operations with exposure and with elevation of the brain from the floor of the middle fossa in one case, and two died following simple mastoid operations without cerebral exposure.

Case 1. In 1916 I did a simple mastoid operation on an adult male, one L. E. Three weeks later, with a profuse middle ear discharge, but with a satisfactory healing mastoid wound, I exposed the dura over the antrotympanum cavity because of persistent temporal and eye pain. I recall a copious discharge of pus from the wound, on a fairly immediate subsequent dressing, with prompt relief of pain and a rapid uninterrupted recovery.

Case 2. At this meeting, eight years ago, I reported "Acute Otitic Temporosphenoidal Abscess, Septicemia, Gradenigo's Syndrome, Acute Surgical Mastoiditis, Acute Suppurative Otitis Media, Frontoethmoidal Empyema, Grippe: Report of a Case with Postmortem Findings."

Referring to the pain in this case, associated with the suppurative otitis prior to mastoid operation on the 19th day, "the most annoying pain was referred to the left temple and left external canthus. Lacrimation and some ocular distress also existed."

"On the 26th day the temperature rose to 102°, there was pain about the left temple and in and about the left eye."

"On the 33rd day there was a homonymous diplopia. On the 40th day paralysis of the external rectus."

"On the 80th day facial paralysis."

"On the 120th day, postmortem, a destructive ostitis of the superior border of the petrous portion of the temporal bone, near the apex was demonstrated, which had produced a concavity three-eighths inches in depth."

Several prominent otologists saw this case and opinions differed as to whether a lesion existed in the frontal or temporosphenoidal lobe, but by no one was there a suggestion made of the consideration of a petrositis as we think of it today.

Quoting further from my report of Case 2, "It is to be noted that there were dural exposures made at the time of the mastoid operation. Theoretically, it seems that it would have been an advantage if such exposures had been made.

"It is my impression, however, that most surgeons feel chagrined at accidental exposures of sinus or dura. Experiences such as narrated in the management of this and similar cases prompt the question as to whether accidental or purposeful exposure of the dura or sinus, either or both, have influenced subsequent events."

What amounts to little more than a dural exposure seems now to have a very proper place in the management of certain cases of petrositis, as is suggested by the management of my first case and third case, and in the experience of others.

Case 3. Miss E. B., female, age 29 years, on whom I did a left simple mastoid operation April 16, 1932, had had a discharging ear following spontaneous rupture for one month following the grippe. Except for profuse discharge and for some loss of weight, there were no other indications of mastoid disease. At this time there was an increasing frontal and parietal headache with inability to sleep.

The mastoid was large with deep cells, nest of cells behind the sinus and cells deeply situated in the tip. No sinus or dura was exposed.

There was complete relief from pain for four days, but pain returned on April 20, referred to the orbit and temporal regions. There also appeared anesthesia and weakness of the fourth and fifth digits of the right hand. During the following eight days the pain remained unchanged as to location, but grew more intense, making sleep impossible without the

use of sedatives. The temperature through this period was slightly above normal, only once over 100°, by mouth.

On April 28, I opened the wound and obliterated a triangular area of cells located above the antrum, by X-ray, and made a large dural exposure over the antrotympanum cavity, elevating the dura and brain over the petrous. No fluid was seen to escape.

The next day pain was much less and confined only to the supraorbital area.

Five days later pain referred to the ophthalmolic branch of the fifth nerve had entirely disappeared and did not return.

Six days postoperative there was diplopia and almost total paralysis of the left external rectus. The fundus was normal.

Patient was discharged on the ninth day after the second operation.

Eight days later the ear was dry and three weeks postoperative the mastoid wound was completely closed.

At the end of eight weeks the diplopia disappeared. The anesthesia and weakness of the fourth and fifth digits of the right hand still remains.

Case 4. L. W., female, age 13 years, first seen June 2, 1932, thought ill with the grippe, had a left acute middle ear abscess which had ruptured. I did an immediate myringotomy.

Her condition was not remarkable for about ten days.

For one week prior to June 21, the patient's nights were progressively uncomfortable. On this day frontal headache, which had been troublesome for a few days, increased; the temperature rose to 103°, with vomiting and an increase in the middle ear discharge. I did a simple mastoid operation June 22.

Just before operation, peeling of the finger tips was noted and the health officer ordered the patient to leave the hospital immediately postoperative. The mastoid, pneumatic in character, was extremely overdeveloped, especially in depth. There was no manifest bone destruction. Occasional cells were filled with fluid pus. No sinus or dura was exposed.

After running a flat postoperative temperature for over a week, the discharge from the middle ear having ceased on the fourth day, there were no remarkable notations made with regards the convalescence except that I thought I noticed a tendency of the patient to shield the left eye from light by voluntarily narrowing the palpebral fissure. This behavior was described by the patient and parents as habit. There was, on July 3, a sudden rise in temperature to 103° and a nagging headache became generalized with paralysis of the sixth cranial nerve. Immediate spinal puncture showed 1300 white cells, a second, 36 hours later, a nonhemolytic streptococcus.

Cultures from repeated spinal punctures for drainage showed increased number of colonies. Intracarotid treatment with Pregl's iodine solution was instituted daily without beneficial effect.

Death occurred July 15 from a generalized meningitis. An autopsy could not be obtained.

I mention the following reasons for bringing this subject to your attention at this time:

1. To attempt to clarify your conception of a condition not at all well understood until very recently.

2. To point out that while this condition is indeed serious, we should exercise our wits in determining when surgical attack of the petrous pyramid should be executed rather than rushing in early with perhaps disastrous results, since there is ample evidence that quite conservative treatment in many cases will suffice.

3. To comfort those of you who may have condemned yourselves for accidental dural exposures in cases which went badly with the thought that it may be probable that those cases terminated fatally, not because of exposures, but because in spite of them.

4. To suggest that you do secondary simple mastoid operations if they seem indicated and that they be done early.

5. To advise not to hesitate to sacrifice the structures of the middle ear in an effort to save the patient.

All of which was given to us in the words of Kuster[11] in 1889, "to open up the bone freely so that it can readily be scrutinized to clear away all disease and so fully to expose the source of suppuration that the pus is nowhere checked in its outflow."

In order to bring the question of infection of the petrous apex up to that date, may I read from a letter I received from Dr. Eagleton recently?

He writes: "I have had a large number of recoveries of cases where the unlocking operation was performed—two of them in which there were micro-organisms in the cerebro-spinal fluid."

So far as my knowledge goes, these two recoveries of cases showing a bacterial meningitis of otitic origin are the first to be reported.

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144 Golden Hill Street.

SUPPURATION OF THE PETROUS PYRAMID.*

DR. SACKS BRICKER, Philadelphia.

This case is reported for two reasons: First, because all the patient's trouble might have been avoided had impacted cerumen been removed from his external canal by an experienced physician. Second, the fact that his symptoms were not typical of those of suppuration of the petrous pyramid.

R. K., male, age 39 years, presented himself on March 12, 1932, suffering with intense pain in the right ear. He gave a history of having gone to a hospital dispensary on March 8 for the removal of wax from the right ear. An interne attempted to remove this wax by instrumentation, causing intense pain and laceration with bleeding at the time of his manipulations. When seen on the 12th, there was a slight discharge of pus from the right ear. The drum was bulging; it was incised and a dry wick placed against the drum. He seemed to improve somewhat up to March 16. The next day there was a bulging of the upper anterior portion of the tympanic membrane. This was reincised and pus under pressure was released. A drain was inserted against the tympanic membrane and changed daily. He was apparently improving during the following week, when on March 26 he began complaining of pain behind the ear. This continued for several days with increasing intensity over the mastoid tip. The posterior superior wall of the external auditory canal showed some evidence of drooping and during the next few days a surgical mastoiditis developed.

On March 30, the patient was admitted to the Temple University Hospital. His blood count was 4,000,000 R.B.C., 10,850 W.B.C., 77 P.M.N., 18 S.L., 4 L.L., Hb. 75 per cent. Urinary findings, except for a faint trace of albumin, were normal. A right simple mastoidectomy was performed. The cortex of the mastoid was unusually sclerotic; the mastoid cells were exenterated; the necrotic tip was removed; cells in the zygomatic root were cleaned out and the dura of the middle fossa was exposed due to the extensive necrosis of the

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mastoid cells in that region. The mastoid was of a large pneumatic type. A rubber tube was used as drainage and the wound closed with Michel clips.

Following the operation the patient ran a temperature of 102.6° F. for two days. The next three days the temperature stayed around 101° F., when on April 6 it rose to 104.4° F. He had a chilly feeling, but no distinct chill. At midnight his temperature had dropped to 97.6° F. There was no Kernig's sign present, nor was there any rigidity of the neck. He complained of a slight cough and on examination of his chest only a few scattered rales were found in the posterior part of the upper lobe of the right lung. A blood culture and blood count were ordered, the count showing 4,250,000 R.B.C., 18,400 W.B.C., 54 P.M.N., 22 S.L., 4 L.L., Hb. 72 per cent.

On April 7, his temperature again rose to 103.6° F. at noon. The purulent discharge from his mastoid wound was most excessive as compared with what it had been previously. Pain on the right side of the head and behind the orbit of the eye was intense. On the morning of the 8th, the temperature dropped to 97.4° F., and by 4 P.M. had risen to 102.4° F. He also complained of an occasional chill. A lateral sinus thrombosis was suspected. A spinal puncture and a Tobey-Ayer's test were ordered. Pressure on the right internal jugular vein produced a spinal fluid pressure of 16 mm.; over the left internal jugular vein 14 mm. Spinal fluid pressure at the time of lumbar puncture was eight mm. and after the removal of ten cc. of spinal fluid fell to four mm. This spinal fluid was clear and gave a count of three small lymphocytes per cc.; sugar 72 mgs. per 100 cc. The wound was redressed, laid wide open and a rubber drain inserted. Although the temperature was of a septic nature, the patient felt unusually well. Reports on his blood culture showed no growths. He was given 20 cc. of one-half per cent neutral acriflavin intravenously and this was repeated the next day. His temperature dropped to normal on the morning of the 9th, and remained so until he was discharged from the hospital on April 13.

The pain over the right temporal region and in and above the right eye which had started on April 7 and was of an intermittent type, by the 17th had become more severe and

continuous in character. There was an occasional diplopia with hazy vision. The lower half of the right side of his face appeared to be somewhat smoothed out, but was of a transient nature. By the 18th the right internal strabismus had become well developed. In coming into my office on the morning of the 18th, the patient was walking toward the left corner of the room when, after a few steps, he swerved like a drunken man and landed on the right side of the room instead. There was no dizziness, nausea or falling sensation. About noontime he experienced a sudden relief from his intense pain in the eye and for the first time since he had been sick his appetite returned, and he said he felt as if nothing was wrong with him except for a good deal of soreness that remained over his eyebrow and in the eye. The discomfort in his eye was still severe enough to interfere with his vision. Right internal strabismus was pronounced.

I was now concerned with the possibility of the case entering the quiescent stage of a suppuration of the right petrous pyramid and had him admitted on April 19, 1932, to Temple University Hospital for further study.

Blood count was 3,500,000 R.B.C., 7,150 W.B.C., 63 P.M.N., five monocytes, 32 lymphocytes and Hb. 70 per cent. His blood chemistry and urine were normal.

The neuro-otologic examination by Dr. J. Winston follows:

"There is a disproportion between the duration of nystagmus and vertigo after turning. There is an appreciable loss of function of the entire vestibular mechanism on the right side. The hearing is somewhat impaired in this ear. There is a more marked loss of function of the left vertical semicircular canals while the left horizontal responds fairly well. The hearing is good in the left ear. These findings are suggestive of intracranial disease. The cerebellum, cerebellopontile angles and posterior cranial fossa appear to be negative. The pathology is probably extradural and in the right parietal region."

Examination of the eye grounds and visual fields was reported normal.

Neurologic report by Dr. N. W. Winkelman: "The patient complains of double vision; this is easily explained by weak-

ness of the external rectus muscle of the right eye, which amounts to practically complete paralysis. There is no weakness of the seventh nerve, the tongue is protruded in the median line and shows little or no tremors. He tells us that the headaches have disappeared. Dr. Nathan has looked into the eye grounds and pronounced them absolutely normal. Prior to this time it is developed that he had a pain back of the right eye.

"He is perfectly comfortable aside from the double vision. Appetite is good, sleeps well and has no general complaints. This represents a case of the so-called Gradenigo syndrome. This condition represents an inflammatory process of the apex of the petrous portion of the temporal bone and amounts to a petrousitis. These patients all get well without any residuals."

X-ray reports by Dr. W. Edward Chamberlain follow:

April 18, 1932. *Region:* Skull, complete study. "The films demonstrate the operative defect in the right mastoid with chronic inflammatory changes in both maxillary antra and with untreated caries destroying crowns of certain teeth. The opinion, which is especially important to Dr. Bricker, concerning the right petrous bone and neighboring regions, is not yet complete. I have asked the patient to come in for further studies, which will be promptly reported."

April 18, 1932. *Region:* Additional films and supplementary report with regard to skull.

"The operative defect in the right mastoid, together with the mucous membrane thickening in the maxillary sinuses and untreated caries in the teeth, have been previously reported.

"We have made a number of special angles of this skull and have found it very difficult to interpret the X-ray appearance, but I am now ready to point out that on the right side, in the region of the suture between the basilar process of the occipital bone and the tip of the petrous portion of the temporal bone there is a definite absence of bone. The bone destruction or absence of bone does not appear to affect the occipital bone or the sphenoid bone, but does definitely involve the tip of the petron. I think it should be borne in mind that

this condition as visualized in these X-rays might possibly be a developmental asymmetry of the petron, but the very important possibility, which is borne out to some extent by the clinical findings, is that of a suppuration in the bone at this part of the right petron."

On April 20, 1932, at a meeting of the Section on Otolaryngology of the College of Physicians, at which Dr. Samuel J. Kopetzky was a visitor, I took the opportunity of presenting to him for an opinion a short sketch of the case with the X-ray films. Dr. Kopetzky pointed out the pathology of the right petrous pyramid and suggested that the patient be operated on. The operation was performed the next day at Temple University Hospital. The mastoid wound was reopened and granulation tissue, together with a few more mastoid cells which were uncovered and some necrotic bone, was curetted until the mastoid looked clean. The operation was carried a step further and a radical mastoid with the removal of the root of the zygoma and a sufficient portion of the glenoid fossa to bring into view the Eustachian orifice was done. Infected and broken down peritubal cells were removed. There were no fistulous tracts found in the middle ear. The opening in the floor of the middle fossa was enlarged due to bone necrosis. With a $22\frac{1}{2}^{\circ}$ "angle-finder" which I devised from a metal applicator and a piece of block tin, finding the point designated by Kopetzky and Almour, between the basal coil of the cochlea, internal carotid artery and Eustachian tube through which to enter the cortex of the petrous pyramid was facilitated. The pyramid was opened with a one and one-half mm. drill and slightly enlarged with a curette. A probe was placed into this opening and very easily entered the pyramid for a distance of about one and one-half inches. The petrous pyramid seemed to be hollow and fearing that I had entered the cranial cavity, I left the probe in the pyramid and through an opening in the middle fossa lifted the dura and brain off the pyramid and was able to see that the probe had not perforated into the cranium. There was no discharge of pus from the opening into the pyramid. This does not necessarily mean that we were not dealing with a suppuration of the petrous pyramid, for it is quite possible that it spontaneously ruptured into the middle ear and it was discharging through the mastoid wound at the time of the very profuse discharge noted as beginning on April 7. The X-ray reports

were further proof that there was a destructive process present and the otoneurologic evidence also pointed to a perilabyrinthian irritation present from an inflammation of the petron. The radical operation was completed and the patient returned to bed.

In the morning following the operation patient stated that his head felt entirely different; that the soreness in the eye and over the eyebrow had disappeared. There was no further pain on the right side of the head and the haziness of vision was clearing up. On the 23rd, patient informed me that he was able to read. The strabismus was barely noticeable. The symptoms began to clear up very rapidly and by the 29th the patient had fully recovered and was discharged from the hospital.

From April 9, the patient's temperature never rose above normal except the day following the second operation. He had an uneventful convalescence, except for a partial loss of hearing in his right ear.

An X-ray check-up by Dr. Chamberlain on Dec. 21, 1932, is as follows:

Region: Special studies of skull for comparison with films of April 18, 1932.

"The present studies comprise stereoscopic projections from the hair line back toward the occiput and from the caudal aspect of the lower jaw toward the vertex.

"The most instructive films are those obtained as vertex projections. When viewed in the stereoscope the films bring out the presence of an unusually large lacerate foramen at the tip of the right petron. This was commented upon at the examination last April and it was difficult to decide what was the significance of this finding.

"Looking back over the case, and comparing the old with the recent films, it appears to me that there was definitely bone destruction at the medial end of the right petron, and that there had been some regeneration of the bone in this region. It furthermore seems apparent that a considerable part of the area of absent bone detail is due not to bone destruction, but to the unusual size of the lacerate foramen."

RESUME.

This case must be considered one of suppuration of the petrous pyramid because of the following facts: *1*. Following an acute mastoiditis, the development of intense deep-seated pain behind the eye, severe pain over the right eyebrow and the parietal region; *2*. The sudden profuse discharge of pus from the mastoid wound and to some degree from the external auditory canal; *3*. The period of low grade sepsis following the mastoidectomy, until the flare-up of the temperature from the seventh to the tenth day after the first operation, when the inflammation in the petrous pyramid was at its height; *4*. The extensively pneumatized development of the mastoid; *5*. The roentgenologic findings showing positive evidence of bone destruction at the medial end of the right petron, and further evidence seven months later that there was some regeneration of the bone in this region; and *6*. The abatement of the pain in the eye and the sudden improvement of the patient following the operation on his petrous pyramid.

In closing, I wish to thank Dr. Matthew Ersner, both for the use of his ward at Temple University Hospital and his assistance at the operation.

3701 N. Broad Street.

FAMILIAL DEAFNESS: A POSSIBLE EXAMPLE.*†

C. C. BUNCH, PH. D., St. Louis.

The complete chain of facts definitely proving the hereditary transmission of deafness is rarely if ever secured. Regardless of this fact, certain types are, on the basis of the evidence in the literature, usually considered hereditary, and when other possible causes have been eliminated, the otologist is compelled to consider heredity the etiological factor even though the actual proof is wanting. For example, much evidence is available which indicates rather strongly that otosclerosis is a disease transmitted by heredity.[1] In fact, many otologists will make a diagnosis of otosclerosis only where the evidence of hereditary transmission is secured.

Another type of deafness usually considered hereditary is that appearing in the children of deaf-mute parents. But these two types of deafness are not easily mistaken. That resulting from otosclerosis is insidious in its onset, appears most frequently in late adolescence or early adult life and quite consistently yields the Bezold triad in the functional tests of hearing. The deafness appearing in children of deaf-mute parents is usually profound and is evident very early in life. But in neither instance is the evidence of hereditary transmission definitely established, for many children born of otosclerotic and deaf-mute parents have normal hearing. Of the two, Tinkel[2] states that in 38 family history charts for cases of otosclerosis "a larger proportion are affected with this defect . . . than suffer from deafness in families of deaf-mutism."

Another type of deafness which is sometimes considered hereditary is that so classically described by Hutchinson, the deafness of the so-called hereditary syphilis. This type should be mentioned in the same category as those above. It may, however, be more rightly called congenital than hereditary.

*From the Department of Oto-Laryngology, Oscar Johnson Institute, Washington University Medical School.

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The accuracy of the term *hereditary* in describing these types of deafness is open to criticism. Genetic studies, such as that of Davenport and others, reveal strikingly the difficulties in the way of the solution of the problem. The use of the term *familial* is perhaps more accurate of description, but it adds little or nothing to our knowledge of the etiology.

A fourth type of deafness of somewhat similar character is less frequently encountered, is recognized with greater difficulty and may be improperly diagnosed even with most thorough study. Dr. L. W. Dean and the writer,[3] in 1923, reported a study of the auditory acuity in three brothers, 8, 11 and 16 years of age, from a family of seven children. The deafness in each case was slight when discovered and appeared to be slowly progressing. The parents were not deaf nor was their marriage consanguineous. The Wassermann tests on the boys and their father gave negative results. No treatment instituted had any effect on the progress of the deafness. The diagnosis was neurolabyrinthitis, etiology unknown. The same study contained a report of similar deafness in two children and in their mother and maternal grandmother. The diagnosis was hereditary neurolabyrinthitis.

Macfarlan,[4] in 1927, under the title, "Identical Hearing in Identical Twins," showed the audiograms secured from tests on middle-aged twin women with this comment: "The hearing rises and falls across the pitch range in nearly exact correspondence one with the other," but he makes no comment as to the probable etiology. Shambaugh and Shambaugh[5] also reported cases of deafness in identical twin women, 31 years of age, and in twin men, 69 years of age. Concerning the deafness in the women, they state that "both gave the functional reactions of primary nerve deafness." Concerning the etiology of the deafness in the men, the writers conclude that it is "also of primary nerve type" and since there was a history of similar deafness in other members of the family, they were led "to question the possibility of some of these cases being the labyrinthine type of otosclerosis."

Rodin[6] in the same year also reported similar deafness in twin girls, 15 years of age. The Rinné test gave negative results and the audiograms showed quite uniform loss for all tones. He concluded tthat the diagnosis was otosclerosis.

This study concerns the hearing of five sisters, 6, 10, 12, 14 and 17 years of age at the time of the first examinations. The father is a factory employe, 42 years of age. The mother is 39 years of age.

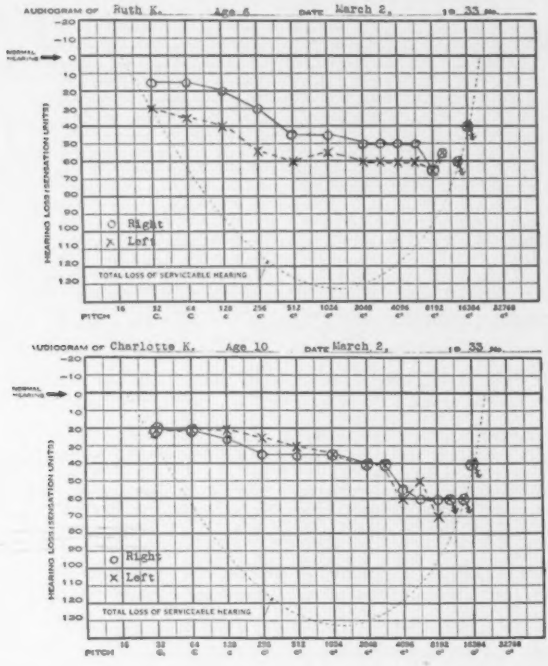
AUDIOGRAM OF Ruth K. Age 6 DATE March 2, 10 33 AM.
HEARING LOSS (SENSATION UNITS)
Right
Left
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF Charlotte K. Age 10 DATE March 2, 10 33 AM.
HEARING LOSS (SENSATION UNITS)
Right
Left
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 1. Audiograms of two sisters, Ruth, age 6, and Charlotte, age 10, at the time of the first examination. Low tones are heard well by both, but the acuity diminishes for tones of higher pitch.

Ruth, age 6 years, and Charlotte, age 10 years, were brought to the Oto-Laryngologic Clinic of Washington University on the advice of Dr. Max A. Goldstein and Dr. Allan Potter. They presented audiograms secured in tests by Dr. Goldstein and his associates. The hearing tests were repeated in the clinic and found to be essentially the same as those secured by Dr. Goldstein. The results of these tests are shown in Fig. 1. The results show that the loss in acuity is bilateral. Low tones are heard well in both ears of each child, and the deafness

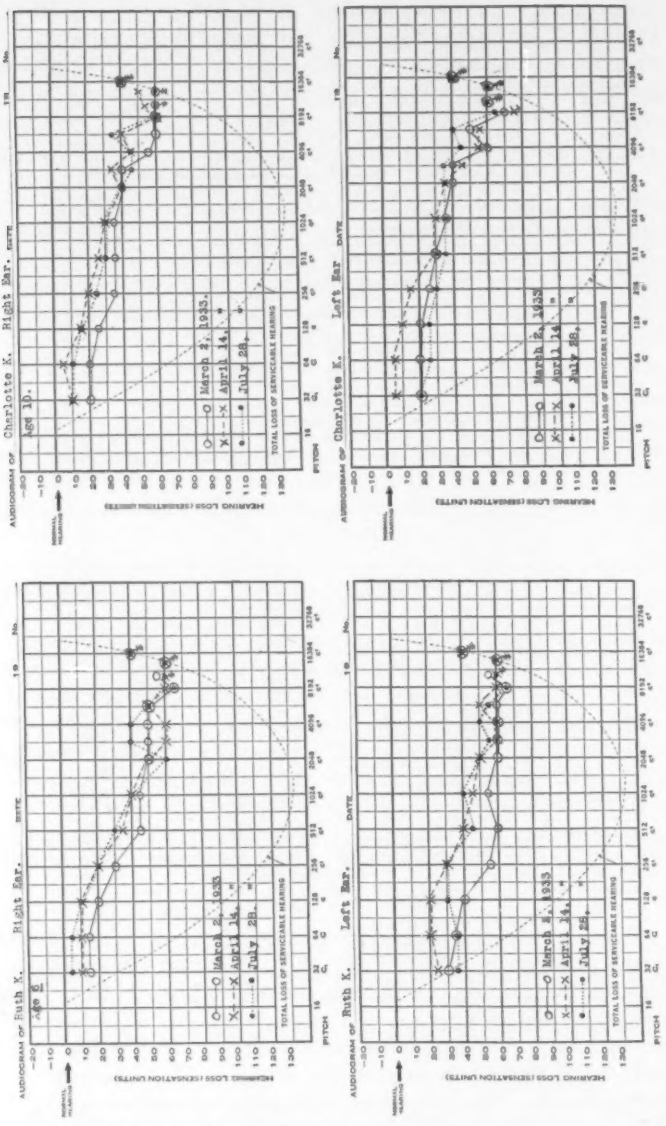

Fig. 2. Audiograms of Ruth and Charlotte showing improvement in hearing on the first test (April 14) after the removal of tonsils and adenoids. The third test (July 28) shows less definite improvement.

increases with the frequency, becoming quite marked for tones of very high pitch. The Rinné test gave the positive result and the Weber test gave no lateralization. The laryngologic examination revealed very bad tonsils and adenoids in each case. A diagnosis of acoustic neuritis was made and it was recommended that the diseased tonsils and adenoids be removed with the thought that the deafness was resulting from some toxic focus, most probably located in the diseased tonsils and adenoids. When the children entered the hospital to have this work done, it was found that the older child had some infection of the maxillary antra and her urine showed four plus albumin, but there were no red blood cells or casts. The Wassermann tests were negative. Both children had their tonsils and adenoids removed, and in the older child antral windows were made for irrigation and to secure better drainage. Their course in the hospital was uneventful and both were discharged in a few days. Six weeks after the first test had been made in the clinic, the children returned for examination and to have the hearing tests repeated in order to determine if there had been any change in acuity of hearing resulting from the removal of the toxic foci. Fig. 2 shows the records of the tests made before and after the operative work, together with the results of a third test made six weeks later. These charts show that the best record was made on April 14, a short time after the operative work. Changes appearing between April 14 and July 28 are too small to be definite. When the second test showed slight but apparently definite improvement, it was considered that the correct diagnosis has been made and that the improvement would continue. But when the results shown by the third test were secured, considerable doubt was expressed.

About this time, the children's mother reported that she thought her oldest daughter's hearing (Anna Marie, age 17 years) might perhaps not be as acute as it should be. She was requested to bring this daughter to the clinic for study. This was done and the results of the hearing tests are shown in Fig. 3. This girl had completed her high school course and was taking an advanced course in business training. The very evident progress she had made in her school work seemed to indicate that her deafness was of recent origin.

When this record was secured, more doubt than ever was expressed as to the accuracy of the original diagnosis in the

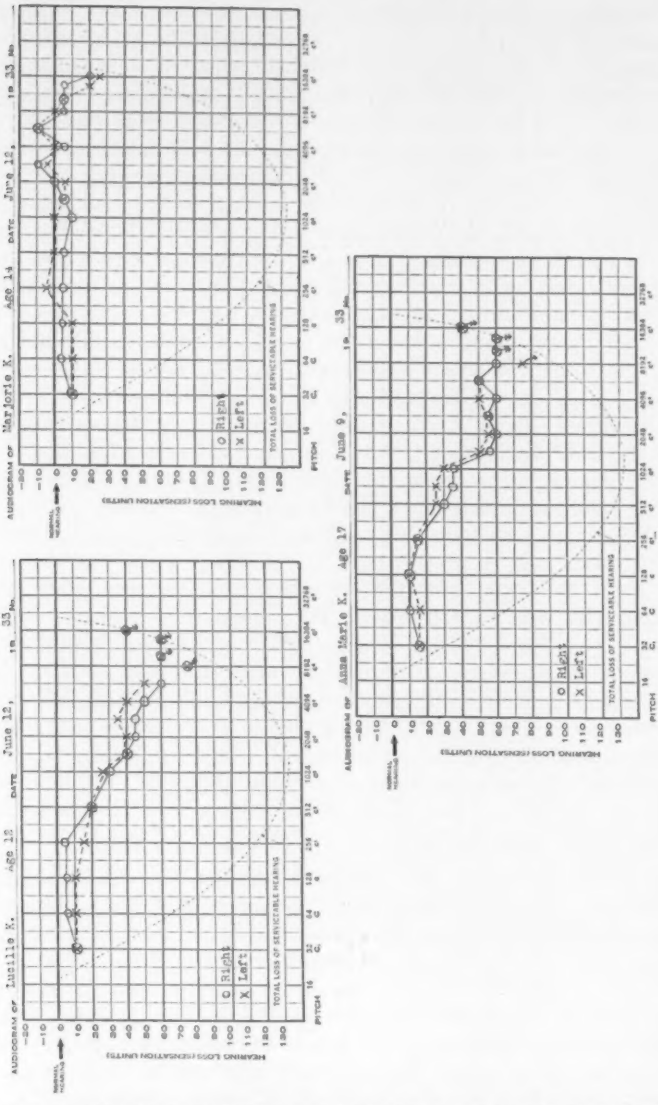
AUDIOGRAM OF Marjorie K. Age 14 DATE June 12, 19 33 No.
AUDIOGRAM OF Lucille K. Age 12 DATE June 12, 19 33 No.
AUDIOGRAM OF Anna Marie K. Age 17 DATE June 9, 19 33 No.
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
O Right
X Left
NORMAL HEARING

Fig. 3. Audiograms of Lucille, age 12; Marjorie, age 14, and Anna Marie, age 17, sisters of Ruth and Charlotte, whose records are shown in Figs. 1 and 2. Note that Marjorie has essentially normal hearing.

case of the two younger children. The records of all three showed the same general type of hearing loss and the question was then raised as to whether or not we were dealing with some form of familial deafness. The mother was asked if there were other children in the family, and replied that there were two more girls, but that their hearing appeared

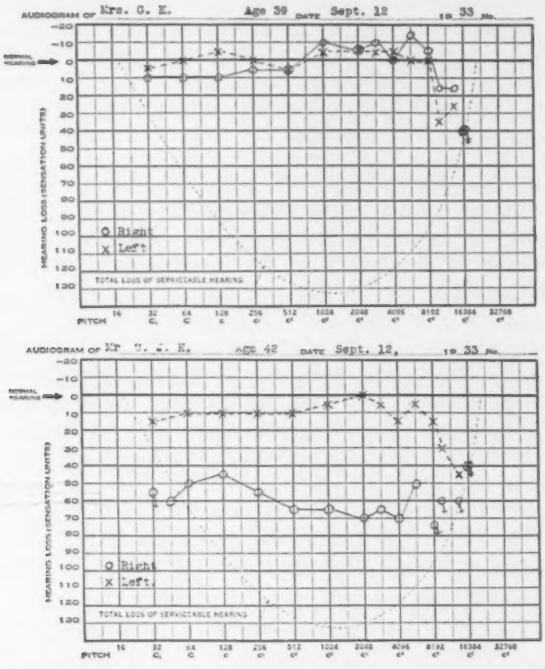
AUDIOGRAM OF Mrs. C. K. Age 39 DATE Sept. 12 19 33 No.
HEARING LOSS (SENSATION UNITS)
O Right
X Left
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF Mr C. W. K. age 42 DATE Sept. 12, 19 33 No.
HEARING LOSS (SENSATION UNITS)
O Right
X Left
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 4. Audiograms of the mother and father of the children whose records are shown in the preceding figures.

to be normal. She agreed, however, to bring them for examination. The audiograms showing the results of the tests of these two are also shown in Fig. 3 (Lucille, age 12 years, and Marjorie, age 14 years). That of Lucille shows in general the same type of hearing loss as that shown by Ruth, Charlotte and Anna Marie. The record of Marjorie, while showing a very slight loss for almost all tones, is practically normal, as is her record for the 4A (phonographic) audiometer.

In order to secure all the information possible as to the probable etiology of this deafness, audiometric tests were done on the father and mother. These records are shown in Fig. 4. The curves in the case of the mother show that the hearing for low tones in the right ear is slightly less acute than that in the left, but differences of this extent and magnitude are frequently encountered and are usually considered insignificant. The loss shown in the father's right ear is accounted for by a history of frequent attacks of otitis media in childhood and youth, and the record showing a rather marked loss for all tones in this ear confirms this history. Neither the father or the mother show losses similar to those secured from the tests of the four children.

Summary: Records secured by means of audiometric tests are given, showing a rather marked bilateral loss of auditory acuity in four girls of a family of five. The similarity of the auditory fields, the lack of a similar loss in records secured from tests of the parents and the absence of a history of deafness in other generations of the family in so far as could be determined have necessitated the use of the term *familial* deafness rather than *hereditary* deafness. The fifth child of the family had practically normal hearing. It is possible that something neither familial or hereditary may be the etiological factor in these cases, but thorough examinations by pediatricians in two of them, internists in one and nasopharyngeal in all four, have not revealed any common etiology. There is no history of common illness, the use of drugs or indications of dietary deficiency. For these reasons the deafness has been called *familial.*

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Scott and Euclid Avenues.

SPONTANEOUS DECOMPRESSION OF A TUMOR OF THE PITUITARY AREA OF THE BRAIN. CASE REPORT.

DR. WM. H. TURNLEY, Stamford, Conn.

There is no region of the body so liable to tumor as the brain, according to Da Costa. During the course of a number of years the necropsies of the Munich Pathological Institute are stated by Bollinger to have shown one tumor of the brain to every 85 necropsies. Hale White's experience is that such tumors are even more common than this, and he estimates them at one in every 59 necropsies. Harvey Cushing, in his recent book, "Intracranial Tumours," says that the pituitary adenomas represent 17.8 per cent of all intracranial tumors, consequently these tumors relatively are not uncommon. At any rate, as with all injuries of the head, it is an important subject, for the frequency of their occurrence, their great fatality, the extraordinary recoveries that are sometimes encountered, the common difficulty in diagnosis, the drama which often leads to or accompanies them combine to surround these cases with a peculiar interest.

A Czechoslovakian woman, age 46 years, presented herself because of a dull ache in the right side and on the top of her head, also blindness of the right eye.

The family history is unimportant as to T.B., insanity, cancer, syphilis or other suspicious conditions. She was married at the age of 23; one child died when three days old, another boy is living, age 17 years. Her menopause appeared at the age of 36, ten years ago.

Her present illness probably began before July, 1930, for at this time she sought relief for her headaches and poor vision. She consulted various doctors here and there, and was finally sent to the Broad Street Hospital in New York, where she was told that she had a tumor of the brain and was completely blind in her right eye. This was in October, 1930,

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and a subtemporal decompression operation was performed on the right side under local anesthesia. This operation relieved her headaches for a short period of time, but gradually they returned and became worse, so she came to this office in May, 1931.

There seemed to be no history of a mental condition except that she was a little forgetful. There was no history of polyuria, glycosuria, sex disturbance or other pituitary symptoms.

Physically, she appeared healthy and had an excellent sense of humor. She had taken on a little weight since the birth of her child. Her blood pressure was 128 mm., systolic, and 70 mm. diastolic, pulse 98 and temperature normal. Her head was rather large in proportion and there was an eliptical area in her right temporal region, 5 cm. by 3 cm., where the bone had been removed. The soft tissue beneath could be felt to pulsate and was tender on pressure. She had a mild chronic ethmoiditis and a small pair of chronic tonsils. The ear drums were slightly retracted, but her hearing was good and the various tests for conduction, reception and vestibular activity were normal.

In the left eye there was a large scotoma obliterating the entire temporal visual field except for the periphery, but with the nasal side of the visual field she could see 20/40 for distance and the No. 3 letters of the Jaeger chart for close work. Her peripheral vision for both objects and color was practically normal. The perimeter showed white visible at 60, blue at 50, red at 40 and green at 30. With the ophthalmoscope, the nerve head appeared small and white with some papilloedema of the nasal side. The pupil reacted to light and accommodation. The extrinsic muscles were normal in action. In the right eye there was complete loss of vision and the optic disc was bluish white and small. There was a slight exophthalmos with a tension of 28 mm., but the movements were good. The pupil was normal in size and reacted to light and accommodation, though it was sluggish and there was no nystagmus.

A neurological examination revealed very little pathology. Reflexes were normal. Babinski test, Rhomberg test, the pointing test and equilibrium tests were all normal.

The blood Wassermann was negative and it was not thought advisable to do a spinal puncture. Urinalysis and blood count were normal.

On July 15, 1931, she came to the office, having had a sudden nose bleed from the right side, at which time quite a bit of dark clots came out, but she felt much better as a result. On examining the nose, there was an opening about ½ cm. in diameter at the site of the ostium of the right sphenoid sinus, which was plugged with blood clot and necrotic material. A blunt probe was inserted gently through the opening until there was felt to be a slight resistance at a point about eight cm. back of the posterior wall of the sphenoid, passing about four cm. above the hypophyseal area. This was done without local anesthesia, for there seemed to be very little sensation. The opening through the anterior wall of the sphenoid was enlarged to about one cm. in diameter and the sinus area curetted gently.

A specimen taken from the lesion in the brain with a pair of forceps consisted of several nodular masses of tissue, each approximately eight millimeters in diameter. Microscopically, the section revealed a rather diffuse cellular tissue composed of small round cells with dense nuclei. In some areas bands and papillae of cells were noted and in a few areas a rosette arrangement. The cells appeared to be chiefly the chromophobe cells of the anterior lobe of the pituitary gland, which showed excessive proliferation. A few basophilic cells were also present. A few large atypical mitotic cells were noted. There were numerous thin-walled blood vessels present throughout the tissue. These sections were similar to several other biopsy slides taken at various times.

Diagnosis: Pituitary adenocarcinoma of the chromophobe cell type.

The nose was cleaned by gentle suction and the probe again inserted with care, while a lateral stereoscopic X-ray picture was taken with the probe *in situ*, which showed evidence of a subtemporal decompression having been performed on the right side, with an area of bone, approximately 5 by 3 cm., removed. The entire shadow of the sphenoid sinus and its roof, including the sella turcica, was absent. The probe ran obliquely upward and backward from the sphenoid sinus to

a point on a line with and 5 cm. above the posterior border of the mastoid in the parietal region. A small metallic foreign body was noted on the right side of the site of decompression (see Fig. 1).

Fig. 1. Lateral view showing probe in situ.

The patient reported several days later, saying that the dull feeling in her head had returned. The sphenoid opening was cleared again of the necrotic material and blood clot, then 20 cc. of lipiodol solution was allowed to run by gravity into the cavity while the patient was lying on her back. Another series of X-ray plates were taken of an anterior-posterior view and lateral view as shown in the accompanying pictures.

She was allowed to return home with no ill effects; in fact, she said she felt much better after the treatment, except for the smell of oil that day.

The patient came in every week for treatment and felt much relieved for a day or so following, then her dull ache

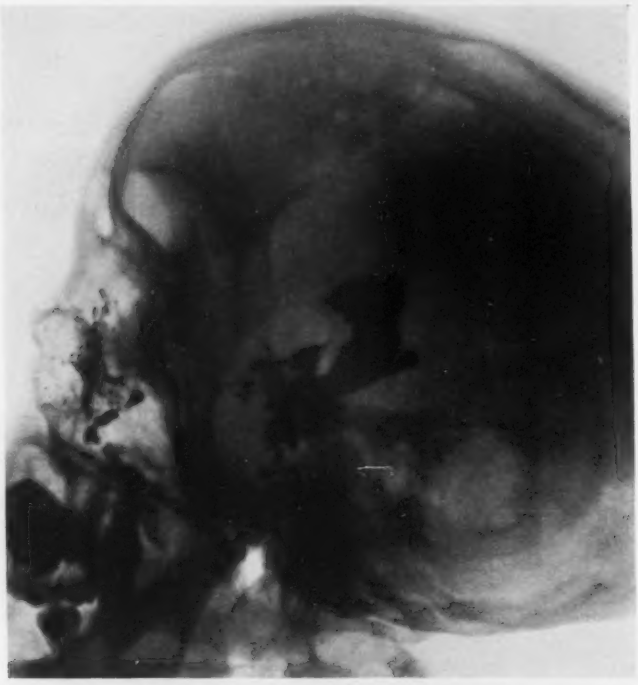

Fig. 2. Lateral view after lipiodol injection.

returned. Her eye condition and general physical condition appeared about the same, though she thought she was improving.

Oct. 29, 1931, she reported for her weekly visit and another X-ray was taken after injecting 10 cc. of lipiodol solution. This showed very little change over the last report, unless the lesion had diminished in size slightly. She was free from headaches, except for a dull feeling over the right eye. The

vision was not improved. The sphenoid opening remained patent and there was no discharge. She slept well, did her house work and in general looked better. Incidentally, she said her memory was improved, so it looked as though a necropsy might be postponed indefinitely.

Fig. 3. Anterior-posterior view after lipiodol injection.

June 16, 1932, she had been seen at regular intervals since the above date and there was no noticeable change in her condition, except a gain of five pounds in weight. The cavity of her brain had not shown a tendency to fill with granulation, though the sphenoidal opening in her nose did close over in

the course of a few days, at which time she developed a slight feeling of fullness in the right side of her head. After this membrane was cleared away and her nose sprayed, she was relieved for another week or so.

On Sept. 13, 1932, she was in the office for the last time and appeared as usual. After being treated she returned home. At 1:30 P.M., the same day, while reclining on a couch, she suddenly had an attack of projectile vomiting, became comatose and died before a doctor could arrive. There were only a few drops of blood from her nose.

It is unfortunate that after this long period of waiting a necropsy could not be obtained. So many of our most interesting cases lack this final diagnosis.

This is only a case report, consequently there will be no attempt to comment on so extensive a subject, but why this patient had not shown more mental symptoms or developed meningitis, a hemorrhage or some other sequellae long ago is difficult to say; however, it bears out the old adage that, "No injury of the head is too slight to be despised or too great to be despaired of."

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DENTIGEROUS CYST OF THE MAXILLARY ANTRUM WITH UNUSUAL VARIATIONS.

DR. JOHN MCGUIRE and DR. H. LEE HARRIS,
Bluefield, W. Va.

A case of dentigerous cyst of the maxillary antrum is reported because of its several unique variations. A general discussion of this rare type of cyst will follow.

REPORT OF CASE.

History: Miss L. M., age 20 years, whose personal and family history were irrelevant, complained of continuous colds over a period of four or five years. She had also observed a slight but slowly progressive swelling immediately beneath her right eye, beginning six or seven years ago. About fifteen months ago, she became aware of a sense of fulness of her right cheek with slight circumorbital pain. A submucous resection of the nasal septum failed to relieve the symptoms and she was told that she had either a "crushed sinus" or a cancer of the cheek bone.

No teeth were missing from the arch. Together with gums and buccal mucosa they appeared to be normal and in excellent condition. There was a marked fulness of the anterior wall of the right antrum in the infraorbital region, causing a moderate asymmetry of the face. Palpation of the hard smooth tumor elicited a slight springiness over the wall. No fistula was discovered, but there was a bulging mass the size of half a small olive, seen in the right middle nares on a plane with the middle turbinate, which displaced both the turbinate and the lateral nasal wall medially (see Fig. 1).

A tentative diagnosis of dentigerous cyst was made. Roentgenograms confirmed this diagnosis in that the cystic wall appeared definitely outlined, the mass being confined to the upper portion of the antral cavity (see Fig. 2). No teeth were demonstrable in this region.

Transillumination of the maxillary sinuses was clear.

The day before operation a needle puncture of the antrum was done, but no fluid was aspirated. Through the same needle, eight cc. of iodized oil was injected. Roentgenograms showed a moderate-sized cyst, confined to the upper half of the antrum, apparently unilocular, having the size and shape of a large olive (see Fig. 3).

Operation: With the patient under general anesthesia, a Caldwell-Luc operation was performed. At point of entrance the maxillary wall was extremely thin, though no dehiscences were found. With wide exposure the cyst occupying the upper half of the antrum was dissected free from the bone. It contained about ten cc. of yellowish chocolate-colored fluid. No teeth were found. Unfortunately, due to firm adhesions, the wall of the cyst was torn in several places and no photograph of it was obtained. Reconstructed, it was similar to the cyst of Harris and Weidlein (see Fig. 4).

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Subsequent to the operation there was moderate bleeding, readily controlled with epinephrine packs in the naris. After five days, the right cheek became markedly swollen, but with the application of cold compresses this condition subsided in three or four days. The patient's temperature curve was practically normal throughout convalecence. A serosanguineous discharge from the antrum persisted for about one month, but gradually decreased until full recovery occurred. Meantime the hard smooth tumor beneath the right eye practically disappeared with restoration of the symmetry of the face.

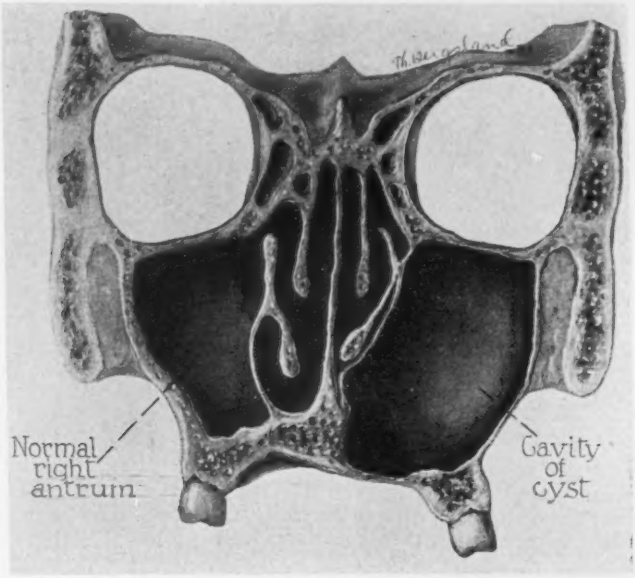

Fig. 1. Normal right antrum and cavity of cyst. Note the thinning of antral wall about the cyst, the depression of the palate, together with compression of the lateral wall and elevation of the floor of the nose. The inferior turbinate is displaced medially and there is a distinct bulging of the cyst beneath its inferior aspect (Harris and Weidlein).

PATHOLOGIC REPORT.

The gross specimen consisted of a roughly ovoid, thin-walled cyst, the size of a large olive. The wall was fibrous with a smooth outer surface. The lining was roughened slightly, the contents having been previously evacuated.

Sections through the cyst wall included a thin lining membrane of stratified epithelium, which varied in thickness. The wall of the cyst was of loose fibrous tissue, being diffusely infiltrated in its entirety by polymorphonuclear leukocytes, endothelial cells and lymphocytes. The first predominated. In some regions of the wall there was found well vascularized granulation tissue, surrounded by small hemorrhagic areas. The diagnosis was dentigerous cyst, seat of subacute inflammation.

Dental cysts comprise two primary types: *a.* the root or periosteal, *b.* the follicular or dentigerous. All varieties of these cysts are known as odontomas and seem to result from a perverted growth of cells in a tooth follicle. The stimulus which is directly responsible for the growth may be congenital or acquired.[1]

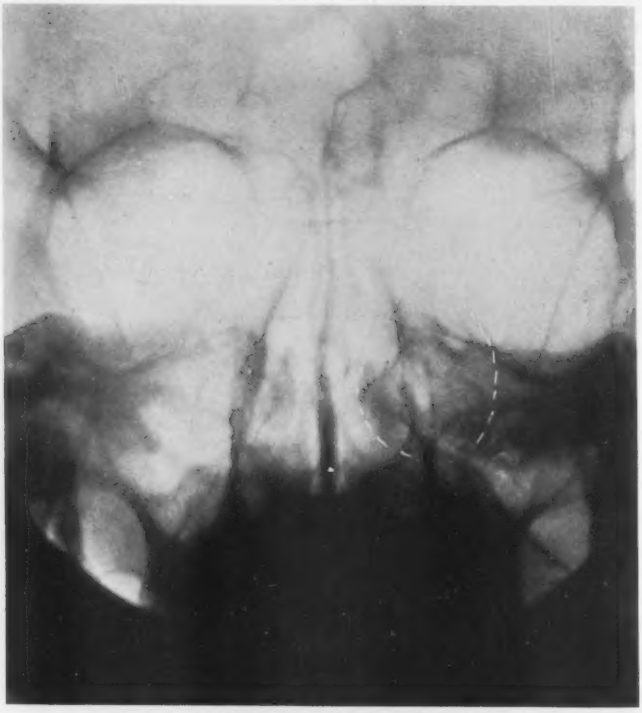

Fig. 2. Posteroanterior view of cystic antrum before operation. The mass of the cyst appears outlined in the upper portion of the antral cavity.

Embryologically, a tooth follicle is composed of the papilla, the enamel organ and the dental sac, the latter arising from the surrounding mesenchyma in the rudimentary jaw[2] and encircling the former component. It is sometimes referred to as the dental anlage.

ETIOLOGY AND OCCURRENCE.

The point of special interest in the foregoing connection, as Wenker[3] observed, is that some of the epithelial cells in the enamel organ cease to function, thereby becoming a source

Fig. 3. Posteroanterior view of cystic antrum following the injection of iodized oil (lipiodol). Note that the cyst has been displaced laterally by force of the injected fluid.

of the "debris epithéaux dentaries" of Malassez,[4] who, in 1885, advanced the theory that all cystic odontomas were derived from this group of cells. Magitot,[5] however, 13 years previously (1872) published the first important work on the subject, holding that cystic odontomas resulted from the development of embryonic dental tissue.

The root or periosteal cyst has its etiology in inflammatory changes in the root membrane due to injury to, or disease of, the root involved. It comprises 80 per cent of all cysts, and is usually found in the upper jaw.

The follicular or dentigerous cyst, of chief concern in this paper, comprises 20 per cent of all dental cysts and as a rule involves the lower jaw.[6] Its occurrence in the maxilla is rare, but it may be found even in the gums or orbit. The cyst is discovered with, or shortly after, the second dentition and usually in patients between 20 and 30 years of age. However, it is occasionally found in connection with deciduous teeth in children and with third molar teeth in persons as old as 65 years.[7]

Fig. 4. Sac of cyst laid open. View of inner wall (Harris and Weidlein).

There are several theories as to the exact origin of the dentigerous cyst, but most authorities concur in the view of Bland-Sutton[8] that it represents an expanded tooth follicle.

COURSE AND CHARACTERISTICS.

The cyst expands progressivly under pressure of its own fluid, sometimes over a long period of years,[1, 9] with consequent absorption of bone and new growth of surrouding tissue.[10] With displacement and absorption of the spongiosa and contiguous tooth roots, the cortex assumes a characteristic eggshell consistency.

The dentigerous cyst of the maxillary sinus is unilocular and may be as large as a hen's egg. Its wall is composed of connective tissue, lined usually with pavement epithelium, with degenerated cells and cholesterol crystals. From the inner wall of the cyst rudimentary, normal or supernumerary teeth may project into the cavity, though *Scudder*[11] *contended that a true follicular cyst may not necessarily contain a tooth, especially if the cyst began its growth early in relation to the development of the tooth involved.* If the cyst contains numerous teeth (Hildebrand[12] found 150 in one cyst), the supposition is that multiple tooth follicles were present. The position of the teeth in the cyst varies.

DIFFERENTIAL DIAGNOSIS.

The history, X-ray and clinical findings usually clear up the diagnosis. A slow-growing painless tumor associated with an unerupted tooth is probably a dentigerous cyst. If no tooth is present, the cyst must be differentiated from a root cyst by history or a test for pulp vitality or by actual operative procedure. If the cyst is painful, diseased teeth or recent accident must be suspected. Rapid growth and pain not explained by injury or localized infection may indicate a malignant condition; and in this connection the general constitutional symptoms, together with cervical adenitis, spontaneous loss of teeth and fetid, blood-stained nasal secretions should be borne in mind.[13]

Maxillary sinusitis is usually ushered in with typical headache and elevation of temperature, engorgement of nasal mucosa, presence of purulent discharge and frequently polypi in the nasal passages.

On the other hand, dentigerous cysts of the antrum, if uninfected, give no appreciable constitutional reaction. A sense of fulness gradually assuming pressure symptoms may be experienced by the patient; also a slight but noticeable asymmetry of the face may occur. Dilatation of the anterior sinus wall, its parchment-like feeling and springiness, and the inner displacement of the lateral nasal wall may clear up the diagnosis. Occasionally the palate is depressed by the expanding cyst and a suspected unerupted tooth is absent from the dental arch. If the cyst has ruptured, there may be a fistula communicating with mouth or nose, usually in the inferior edge of the canine fossa.

The roentgenogram is indispensable in the differential diagnosis of antral conditions, as was shown by Cooper[14] and Feldman[15] and others.

Transillumination may or may not show a cloudiness of the antrum.

As demonstrated by Harris and Weidlein,[1] needle puncture of the sinus, with aspiration of the cystic fluid and the injection of iodized poppyseed oil 40 per cent (lipiodol), with roentgen study, will give valuable information as to the contents of the sinus and the shape and size of the cystic mass.

TREATMENT AND PROGNOSIS.

In the matter of treatment, nothing but a radical operation will prove of the slightest value in these cases. Freudenthal[16] reported the case of a woman, 30 years of age, whose dentist had drilled a hole into the superior maxilla and irrigated the antrum frequently over a period of two years without relief. A radical operation was finally performed by a rhinologist, a dentigerous cyst removed and complete recovery noted in two weeks.

Leon Harris[17] stated that incision and drainage of the dental cyst offer only temporary relief, that recurrence is inevitable, due to the fact that the cyst membrane secretes and in time refills the sac. If the tooth follicle is removed, the cyst will never recur. It is also of interest to note, as Dorrance[18] pointed out, that after a radical operation that part of the bone which has been involved and thinned by the distention of the cyst returns to its normal contour and density.

Of the radical operations, either the Denker or Caldwell-Luc is the one of choice. The prognosis is excellent.

CONCLUSIONS.

A case of dentigerous cyst of the maxillary antrum of rare type is reported because of its several unique variations. Operative procedure is of great importance.

1. Though unilocular, the cyst filled only the upper half of the antrum; by pressure and absorption it had caused a definite bulging of the superior lateral nasal wall and a marked asymmetry of the infraorbital anterior wall of the sinus.

2. The cyst contained no teeth, not even of a supernumerary nature, which seems to confirm the contention of Scudder[11] that a true follicular cyst may not necessarily contain a tooth.

3. There were neither bony dehiscences nor fistulous tracts discovered.

4. This cyst, judging from the history of the case and the fact that dental cysts usually have their origin from a tooth follicle at the time of second dentition, was doubtless in formation over a period of eight or ten years.

5. The Caldwell-Luc or Denker operation is the one of choice in cases of the type above reported.

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208 Bland Street.

POST-GRADUATE COURSE IN OTOLARYNGOLOGY.

The Washington University School of Medicine, of St. Louis, offers an intensive four weeks' course in otolaryngology. The course begins June 18, 1934. It is desired that only qualified practitioners in otolaryngology apply for admission. The purpose of this course is to bring to the attention of the group the more recent advances in otolaryngology. Particular attention will be given to the basic sciences connected with the specialty. The physics, the pathology, the anatomy, the physiology, the embryology, the bacteriology and the cytology will be studied. The influence of nutrition and atmospheric conditions upon the sinuses will be considered. The work in these sciences will be given in the laboratories of the Oscar Johnson Institute.

The clinical work will consist mainly of a study of the relationship between diseases of the ear, nose and throat and systemic disease. Otolaryngologic diagnoses, including endoscopic, will be stressed in clinical conferences and on ward walks. Most of the operative work will be given in the anatomical laboratory. There will be clinical and laboratory demonstrations of allergy of the nose and sinuses. Attention will be given to the care of the deaf child and to speech defects.

A limited number of otolaryngologists who desire to specialize further along particular lines in otolaryngology are, on completion of this regular graduate course, permitted to continue with special work if arrangements are made with the member of the faculty in charge of the particular branch of otolaryngology in which they are interested.

For information, write to Dr. L. W. Dean, School of Medicine, Washington University, St. Louis.

TONSILLECTOMY ON A BLEEDER; TRANSFUSION; ACUTE GANGRENOUS APPENDICITIS; PERITONITIS; RECOVERY.*

DR. HAROLD HAYS, New York.

I am presenting this interesting but simple case to you because it brings up a number of problems which are of vital interest to all of us.

Mr. D. presented himself to me with certain definite symptoms which indicated the necessity for the removal of his tonsils. He was of athletic build and had indulged in athletic activities all his life. His general physical condition was excellent. He gave a history of being a bleeder on two different occasions. In 1919, he received an injury to his nose which resulted in a severe hemorrhage, which stopped only after an injection of horse serum, from which he had a severe reaction. In 1929, he had a tooth removed, after which there was a severe hemorrhage which did not stop for three days.

A test of his coagulation and bleeding time taken in my office showed that it was prolonged to seven minutes. He was directed to take a calcium preparation for one week and then to return for a further test. The second test showed that the coagulation and bleeding time had been reduced to less than five minutes. I suggested that he wait another week before being operated upon, but he was anxious to have the operation performed and so he was admitted to the Park West Hospital the following day. A further test of his blood showed that the coagulation and bleeding time had been reduced to four minutes.

A simple and uncomplicated tonsillectomy was performed with practically no bleeding, but a severe hemorrhage from both sides began three hours later. This was uncontrollable by ordinary methods of procedure. At 9 o'clock that evening a transfusion was performed, whereupon the bleeding ceased immediately.

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*Presented at the clinical conference of the Park West Hospital, Nov. 20, 1933.

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The patient was in good condition for two days, when he began to complain of abdominal pain. Examination of his abdomen gave no indication of any trouble and instructions were given to clean out his bowels with cathartics and enemas on the theory that the pains were due to irritation of his intestinal canal from the blood he had swallowed.

On the third day he had a rise in temperature to 103° and again complained of the abdominal pain. A hypodermic injection of morphine relieved him considerably, but on the following day, when he was supposed to go home, his temperature was 101°, and he was advised to remain in the hospital although the abdominal pains had disappeared. He refused to remain, however, and so signed a release from the hospital.

A few hours later, after his return home, pains in the abdomen began again and the family physician was called in to see him. His first thought was that the man had a thrombophlebitis and three men prominent in the medical profession were called in consultation, none of whom could make a diagnosis of the most possible obvious condition—acute appendicitis—until 12 hours later, when he was rushed to the hospital and operated upon for an acute gangrenous appendicitis with peritonitis. The patient had a stormy time of it but eventually recovered.

This case presents a number of interesting points. Primarily, of what value is a routine coagulation and bleeding time test? Secondly, of what value is the administration of calcium lactate before tonsil operations? It is a routine procedure in our hospitals to make these tests, but the consensus of opinion is that, unless a blood platelet determination is made, no one can tell whether a patient is a bleeder or not. You will note that I have called this patient a bleeder instead of a hemophiliac. There was no history of hemophilia in the family and I am convinced that there are certain patients who are definitely bleeders and that we should pay more attention to the previous history of bleeding in such cases. Our routine tests will not give us the proper information. Such a patient would have been far better off if a transfusion had been performed before operation or at least the precaution may have been taken to have withdrawn an ounce or two of blood and to have injected it into the buttocks.

The second point of importance is the relationship between the development of the appendix infection and the tonsillar condition. No culture to determine the organisms in the tonsils nor in the appendix was taken. If the appendicitis had developed while the tonsils were still intact, one may assume that there was a definite relationship, for one has often seen an appendix inflammation light up after an acute tonsillar infection. The appendix condition in this case, to my mind, was purely incidental or may have arisen from the fact that the intestines were inflamed as a result of the amount of blood swallowed. However, one should pay more attention to abdominal complaints in patients who have had nose and throat operations recently performed and realize that a real serious condition may be present.

133 East 58th Street.

AN INOPERABLE CASE OF CERVICAL LYMPHADENITIS, CURED BY ELECTRO-SURGICAL TONSILLECTOMY.

DR. LEWIS J. SILVERS, New York.

Mrs. A. F., age 31 years, wt. 125 lbs., B.P. 115/85, Hb. 60 per cent, delayed clotting time, idiopathic purpuric lesions occurring spontaneously showed evidences of a blood dyscrasia. Infantile paralysis at the age of six years left her with a partial left hemiplegia. Bled very freely on cutting of skin and noted ecchymotic spots on slightest trauma. Gradual enlargement of the left cervical nodes for the past seven years. Frequent sore throat caused the patient to visit many otolaryngologists. All saw the advisability of tonsillectomy, but on account of the possibility of a tuberculous cervical lymphadenitis, no one thought it feasible to operate. Considering this an inoperable case by means of the orthodox surgical operation, her family doctor referred her to me for electrosurgical tonsillectomy.

A physical examination of her throat revealed large, vascular and diseased tonsils. A large quantity of cheesy detritus was expressed from both tonsils. A large matted mass about the size of a hen's egg occupied the area at the angle of the left mandible. Electrosurgical extirpation was begun on March 6, 1930. A cutting current was first used to incise the abscessed tonsil. The smaller vessels and the lymphatics were sealed simultaneously by the cutting current, thus avoiding any extension of the infection. Coagulation was accomplished through the initial incision. Fulguration was avoided here because of the possibility of sealing the apertures for drainage. Following the second coagulation treatment the mass in the left cervical region began to disappear. A short sharp spurt of bloody, foul-smelling discharge issued from the left tonsil when a large piece of coagulated tonsil broke loose. The patient immediately noticed marked relief from the pre-

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vious tenseness of the skin of the neck and the difficulty in deglutition. When the patient returned for her third treatment on the left side the cervical lymph nodes were practically normal in size. The treatments were continued, however, employing both the biterminal and monoterminal endothermy currents until the complete extirpation was accomplished at the end of ten treatments.

This case I consider worth reporting from a standpoint of inoperability, both from the tuberculous phase as well as that of blood dyscrasia. Of the ten surgeons this patient had visited, all agreed on the necessity of tonsillectomy, but none was ready to perform the operation in the orthodox fashion. The electrosurgical technique is especially well adapted to this type of case.

1050 Park Avenue.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Boston, Mass., Sept. 16, 1933, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-nine candidates were examined, of which thirteen were conditioned or failed.

The Board will hold an examination in Cleveland, Ohio, June 11, 1934, during the meeting of the American Medical Association. Also early in the summer (date is not set) an examination will be held in Butte, Mont., in connection with the meeting of the Pacific Coast Oto-Ophthalmological Society. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for proper application blanks.

INVOLVEMENT OF LARYNX IN AGRANULOCYTIC ANGINA. REPORT OF THREE CASES.

DR. MORLEY T. SMITH, New Rochelle, N. Y.

During the past few months, the writer has seen three consecutive cases of agranulocytic angina with extension to the larynx. Judging from the literature, involvement of the larynx is rather uncommon, especially in this country, some ten cases having been reported since 1922, while during the same period, 22 cases were reported in the foreign literature. In these three cases the original lesion was in the pharynx, with extension to the larynx. In two of these cases, a provisional diagnosis of diphtheria had been made, and one case had been given diphtheria antitoxin.

Agranulocytic angina is a recent clinical syndrome that is quite common, and was first described some 12 years ago. If it existed prior to this time, it apparently was not recognized, and a great many clinicians and laboratory men of large experience think it is a new syndrome. A search of the literature shows that involvement of the larynx, with its most distressing effects, is not common.

W. Schultz,[1] in 1922, gave the first description of agranulocytic angina. He reported three cases in women between 38 and 61 years of age, in one of which the larynx was affected. Again, in 1925, he reported two cases of laryngeal involvement, one with necrosis, and the other with inflammatory edema of the upper portion of the larynx. In 1925, W. Schultz and L. Jacobwitz[2] reported two cases, one with involvement of the epiglottis and upper portion of the larynx, and the other with involvement of the epiglottis and aryepiglottic fold. In the same year, I. Zadek[3] reported a case with necrotic laryngitis. Aubertin and Levy,[4] in 1928, summarized the cases from the literature, and found the larynx involved six times in 32 cases, and the larynx was found involved nine times in 24 necropsies. Fitz-Hugh and Comroe[5] summarized, in 1933, the cases since 1927, when the condition

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was first recognized at their clinic, and there was only one case involving the larynx, reported by Rose and Houser.[6] Freckner[7] reported one case in 1929, with extensive edema extending to the larynx, which necessitated tracheotomy, the only one mentioned in the literature requiring tracheotomy. J. Chalier[8] and associates in their studies found the larynx involved in 16 per cent of the cases. Conner, *et al.*[9] report one case in which the larynx, down to and including the vocal cords, was markedly edematous and was covered with a yellow exudate. Kastlin[10] reviewed 43 cases from the literature and states the larynx was involved in five. Geschelin[11] reports a case in which postmortem showed involvement of the larynx. Haken[12] reported a case in a girl, age 8 years, with pseudomembranous inflammation of the larynx.

Other cases with laryngeal involvement have been reported by various authors, where the involvement varied from redness to others with edema and necrosis of the laryngeal structure in various parts.[13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32]

Case 1: White, female, age 39 years. This patient was referred to the hospital by her physician, with a clinical diagnosis of diphtheria. She complained of a sore throat, dyspnea and dysphagia.

Past History: Frequent sore throats.

Present Illness: Onset four days previous, when patient complained of a sore throat, accompanied by fever and general malaise, pains in the neck, and chilly sensations. On the day previous to admission, she was seen by her family physician, who administered diphtheria antitoxin. A blood count had not been done.

Physical Examination: Patient appeared very acutely ill. Temperature, 102.8°; pulse, 142; respiration, 38. There was marked respiratory embarrassment. The cervical glands were swollen and tender. The tongue was coated, breath foul, and both tonsils showed extensive ulceration, and were covered with a grayish pseudomembrane which extended to the adjacent soft palate and onto the lateral pharyngeal wall. Further examination, with the laryngeal mirror, showed this ulceration extending on the right side from the lateral pharyngeal wall to the right aryepiglottic fold. The larynx showed

marked edema and only a slit for an airway remained. A diagnosis of agranulocytic angina was made and an immediate tracheotomy was done to relieve her great distress due to respiratory embarrassment. Immediately the tube was inserted, her color improved and the distress was greatly diminished. A blood count showed 750 white blood cells per cmm., with 90 per cent lymphocytes and 10 per cent polymorphonuclears. Patient was given 10 cc. pent-nucleotide in each buttock. Smear from the throat showed a few Vincent's organisms and many Gram negative and positive cocci. The throat culture showed an occasional colony of hemolytic staphylococci and no diphtheria bacilli. The patient expired 16 hours after admission. A postmortem was not obtained.

Case 2: White, female, age 45 years; was referred to the hospital with a diagnosis of tonsillitis and possible peritonsillar abscess.

Chief Complaint: Sore throat, dysphagia, fever and general malaise.

Past History: Irrelevant.

Present Illness: For several days, she had sore throat and some fever. Her symptoms rapidly became worse, and on admission to the hospital she complained of feeling chilly, with an aggravation of all her local and general symptoms.

Physical Examination: When first admitted, her temperature was 103.2°; pulse, 130; respiration, 22. The following day, after a transfusion, her temperature reached 105.4,° and the following morning, when the writer first saw her, it was 101.° The patient appeared very acutely ill and toxic, with marked dyspnea. There was fairly marked swelling of the left tonsil and adjacent soft palate. There was a slough over the left tonsil, on removal of which the area bled quite freely. The laryngeal examination showed a fairly extensive edema of the left aryepiglottic fold and the left arytenoid and interarytenoid space, and the cords were seen with difficulty, due to the edema. This case did not show any ulceration in the larynx, as was noted in Case 1. The blood count taken the day before the writer saw her showed 3,760,000 red blood cells per cmm. and 500 white blood cells, with 100 per cent lymphocytes. The following day, her total white cell count was 150, with 100 per cent lymphocytes.

Treatment: In order to relieve her terrific respiratory distress, a tracheotomy was done shortly after she was seen, which gave her immediate relief and made her inevitable exit a little less distressing. A postmortem was not obtained.

Case 3: White, female, age 40 years; was first seen at her home in consultation with her family physician, who had made a tentative diagnosis of diphtheria. He stated that he had first treated her about ten days previously for what appeared to be an acute tonsillitis and head cold. During the past 24 hours her symptoms had been greatly exaggerated, and when seen she had very labored breathing, sore throat and dysphagia, and was unable to breathe lying down. She was immediately hospitalized.

Past History: General health had been good, not subject to upper respiratory infections.

Physical Examination: Rather obese woman, appearing acutely ill. The respirations were very labored and she was cyanotic. Temperature, 105°; pulse, 120; respiration, 35. The breath was foul, the tonsils and soft palate were covered with a dirty gray exudate, beneath which there was superficial ulceration. These parts also showed considerable edema. Examination with the laryngeal mirror revealed that this same exudate involved the epiglottis, the right aryepiglottic fold and the arytenoids and interarytenoid space. There was extensive edema of the glottis, only a very narrow slit remaining for an airway. Blood count showed 77 per cent hemoglobin, 3,690,000 red blood cells per cmm., 550 white blood cells, with 100 per cent lymphocytes. Urine showed large amount of albumin, 1.8 per cent sugar, and many granular casts. A tracheotomy was done immediately to relieve her desperate respiratory symptoms. Following this her color immediately improved and she became quiet. While on the table she was given 500 cc. of blood. She expired 16 hours after admission. No postmortem was obtained.

SUMMARY.

1. A routine examination of the larynx should be made in all cases, and in those showing extensive involvement of the larynx, either from ulceration or edema, a tracheotomy may be necessary to relieve their respiratory distress.

2. In all acute throat infections, a complete blood count, in addition to a throat culture, should be done immediately. Two of these cases had been diagnosed as diphtheria, and one had antitoxin administered. It is fairly easy to mistake some of these with a pseudomembrane, for diphtheria, but the white cell count will make the differentiation possible.

3. Agranulocytic angina usually occurs in women of middle age.

4. When the white cell count drops to 1,000 cells per cmm. or less, the outcome is almost invariably fatal.

5. Blood transfusions do not seem to have any beneficial effect. Some authors report good results from pent-nucleotide intravenously, but in most of these cases the white cell count was over 1,000 per cmm.

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421 Huguenot Street.

PEANUTS, NEEDLES AND SAW-HORSES.*

DR. FREDERICK T. HILL, Waterville, Me.

This is an informal discussion of several recent cases, some of which may serve to illustrate certain principles, of varying importance, and some may be amusing, if not instructive. As there is so little relationship between the cases, it is obvious that no formal conclusions from the whole series can be arrived at.

An infant, age 2½ years, had never seen a cat, but was very fond of peanuts. Encountering his first cat, while having a mouthful of peanuts, he gasped and choked, undoubtedly aspirating a bit of peanut. Soon he became ill, ran an irregular fever, had croupy breathing and cough, and lost weight. He was treated by the family physician for bronchitis. No information regarding the peanut episode was volunteered by the parents, nor did the physician incorporate inquiries regarding foreign bodies in his history. On the thirty-eighth day after aspiration, the infant was extremely ill. Temperature was 104°. There had been marked loss of weight. At this point the father recalled the incident of the peanut, whereupon the physician immediately sent the child to the St. Mary's Hospital in Lewiston, where X-ray and physical examinations showed a complete obstruction to the bronchus of the right inferior lobe. The child was referred to me the same day and, one hour after admission to the Thayer Hospital, a bronchoscopy, without anesthesia, was performed. The findings were typical of a marked vegetal bronchitis. The tracheal and bronchial mucosa throughout was inflamed and there was a large amount of foul smelling secretion coming from the right main bronchus. This was filled with easily bleeding granulations just beyond the branch to the middle lobe. After removing these with forceps, an irregular portion of peanut was seen below and removed.

Five hours later there was evidence of glottic obstruction, substernal and intercostal retraction, but no cyanosis. Tra-

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cheotomy was immediately done. This was somewhat hampered by a large thymus gland, which obtruded into the field and required retraction. This recalled to mind that during the bronchoscopy the trachea had seemed compressed from before backwards. Special day and night nurses were put on and every attempt made to keep the airways clear. For the first 24 hours we seemed to be accomplishing something, although it was necessary to remove the tube and clear the trachea below with suction about every three hours. The second day it became increasingly difficult. Twice within a period of three hours I had to hurriedly insert a bronchoscope and aspirate secretion which had flooded over into the left lung. One and a half hours later another attack of obstruction proved fatal. I was not able to obtain permission for an autopsy, but was allowed to make a postmortem endoscopic examination. Although the bronchi were carefully searched, no further foreign bodies were found. The findings, except for a generally inflamed mucosa, were uninteresting until the bronchus to the right inferior lobe was entered, when frank pus was encountered, welling into the opening of the bronchoscope. Here, of course, we had a case of lung abscess and drowned lung, secondary to a vegetal bronchitis. It is interesting to note that the child's temperature came down after the removal of the peanut, despite the continued pathology in the lung.

The lesson in this case, of course, is that the physician should always consider the possibility of foreign body aspiration in every pulmonary case in a child. It would have been better on my part to have done a tracheotomy at the time of bronchoscopy, as it was obvious that it would be required. Thirty-eight days is a long time for a peanut to remain in the bronchus.

Contrast this with another case occurring just previously. A girl, age 5 years, while running, with her mouth full of peanuts, choked and strangled. That night she became croupy and developed a cough and fever. The following day her physician referred her for bronchoscopy. X-ray of her chest was essentially negative, but physical signs pointed to obstruction in the right main bronchus. Several fragments of peanut were removed at bronchoscopy. She had some febrile reaction for two days, which cleared up, and she was discharged

well on the fifth day. In this case the age of the child made a big difference, as the danger varies inversely with the age, but the prompt recognition of the situation by the attending physician resulted in early removal of the offending peanut and recovery.

Sometimes the family physician may recognize the situation and yet not be sufficiently conversant with the problem involved to avoid getting into difficulties. A boy, age 4 years, swallowed a nickel. He was taken to a physician in a neighboring town who has his own X-ray equipment. With this he was able to localize the foreign body in the upper esophagus at the cricopharyngeus. It appeared easy to him, so he had the boy etherized there in his office and attempted to introduce a proctoscope in an endeavor to remove the coin. While this was unsuccessful, he was lucky at that, or perhaps the boy was, for, except for considerable trauma to his pharynx, no great damage had been done, when he was brought to me about an hour later. After insertion of an esophageal speculum, without anesthesia, the nickel was quickly removed and no untoward results occurred. I fear that it still looks so easy to the doctor that he will try again with the proctoscope if he has the opportunity.

Another foreign body case in a child proved difficult of interpretation from the X-ray. There was no history to help one out and conjectures ranged from a medal in those more religiously minded, to a simple lead disc. On removal it proved to be the cover to a toilet seat, doll size.

Two needle cases may be of interest. The first was a young "lifer," a convict in the state prison. While sewing a patch on his pants, he attempted to reach forward for something while holding the needle in his mouth. He slipped and so did the needle. Attempts to remove it from his throat only resulted in making matters worse. The prison physician summoned a consultant. When he was unable to locate the needle he had the young man taken to the Knox County Hospital for X-ray examination, afterwards bringing the patient and X-rays to me. Mirror examination revealed nothing, although from the X-rays it would seem that the needle was in the hypopharynx. He was admitted to the Thayer Hospital, where additional X-rays showed it in the same position. Direct examination was done under local anesthesia and the hypo-

pharynx and upper esophagus carefully explored. No foreign body could be found, but, on the posterior pharyngeal wall above the level of the pyriform sinuses, a small punctured wound could be located. Probing this, I thought that I felt the sensation of a foreign body. Attempts to insert a forcep and extract this were unsuccessful. More X-rays were then taken, which showed the needle to have been moved upward about half the width of one vertebra. Later that same evening the patient's throat was reanesthetized. A small horizontal incision was made over the location, as shown by the X-ray, and after some search the needle was found among the muscle fibers and removed. After a few hours rest the patient, with his guard, was discharged to return to the prison, at 1 A.M. Convalescence was uneventful.

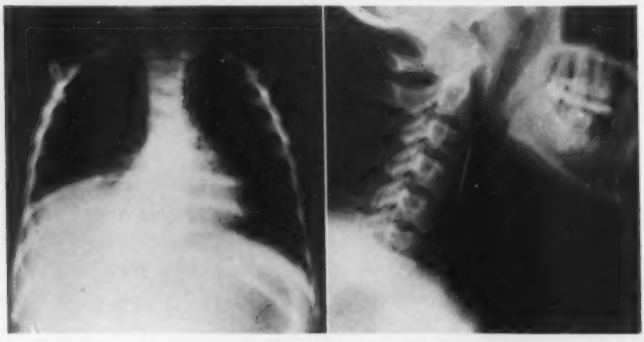

Fig. 1. X-ray of case with peanut in right bronchus 38 days, showing obstruction of bronchus to lower right lobe.

Fig. 2. Lateral view, showing needle in posterior pharyngeal wall.

It is interesting to note that the needle had penetrated the esophageal wall below and turned and gone upward anterior to the vertebrae. The punctured wound was below the actual location of the needle. This indicates the effect of blind attempts at removal.

Here I question the method of approach. One would almost feel safer in attacking this from the external route, as reported by Iglauer at the last meeting of the American Laryngological Association. Circumstances made the internal route the one of choice in this case. Due to prison regulations we

had to return this man within 24 hours. Then we already had an esophageal wound and the foreign body had been felt through it. It was considerably higher than in Iglauer's case and I felt that there was less likelihood of a descending infection ensuing at this point. And a needle is a mighty hard thing to find in any sort of tissue.

The moral in this case is obvious, that no blind attempts should be made to remove a foreign body in the throat, especially a pointed one, as it may be converted from a simple problem into a tremendously complicated one. And one should be prepared to follow and remove the foreign body from any possible point that it may migrate to.

Contrast this with another needle case a little later. A man of 60, while eating oatmeal, felt something sharp lodge deep in his throat. This was so painful that he made no further effort to continue his breakfast, but consulted his physician, who immediately referred him to me. Mirror examination showed an inflammatory swelling and retained secretion in the pyriform sinuses, Jackson's sign of an upper esophageal obstruction. X-ray showed a long thin foreign body in the upper esophagus. Esophagoscopy was done as soon as possible. After considerable search among the mucosal folds in the cricopharyngeus, an ordinary sewing needle, slightly bent, was found and removed. Convalescence was uneventful except for the patient's determination to sue the company manufacturing the cereal. This subsided somewhat when it was pointed out that the foreign body more likely came from the clothing of the housewife who prepared the oatmeal. The obvious lesson here is that cooks should not have loose needles or pins sticking in their dresses.

We are all careful to examine the faucial and lingual tonsil regions in cases of sharp foreign bodies. One case recently taught me to look even more carefully in the mouth. A boy, age 10 years, obviously frightened and apparently in pain, was rushed to my office by his panic-stricken sister. The story was that he had been making a toy airplane and had swallowed a part of a common pin which he had had in his mouth. He could feel a sharp pain in the left side of the neck just behind the larynx. Examination of his throat and larynx revealing nothing, he was sent to the hospital for X-rays. Complete X-rays from the throat to the tubes showed no sign

of foreign body. Instructions were given to watch the stools and to give no cathartics, and to report later. Just before they reached home the boy felt something sharp on his tongue and, inserting his fingers, removed a half a pin which was reposing between two molar teeth. He then remembered that he had placed this pin there, for some unknown reason. Later, not finding the pin in his mouth, he concluded that he had swallowed it. Hereafter I shall look more carefully around the teeth in this type of case.

The last case concerns a man, age 63 years, who was sawing wood on Labor Day. The stick of wood broke and he was thrown forward, striking the front of his neck across the sawhorse. He got up and started to saw more wood when he found his eyes were closing. He managed to get to his neigh-

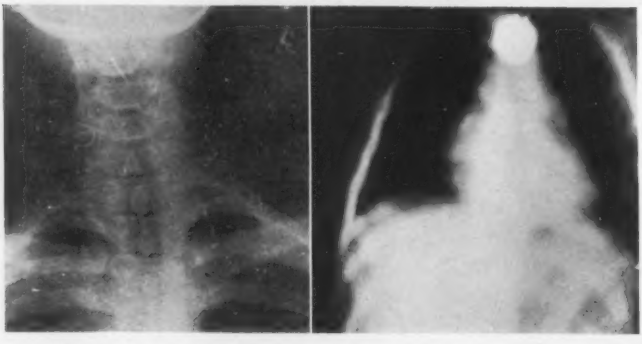

Fig. 3. Anterior-posterior view of same.

Fig. 4. X-ray showing foreign body, doll's toilet-seat cover, in esophagus.

bor's, who took him to the Gardiner Hospital. The attending physician called me. When I arrived there about an hour later, he had marked subcutaneous emphysema extending from his forehead down over his chest and his arms to the wrists. There were marked crepitus over all this area. Both eyes were tightly closed and he was breathing with a great deal of difficulty. His voice could only be described as "squeaky." Laryngeal examination showed marked edema of the arytenoids and glottic swelling, although edema of the epiglottis prevented a view of the anterior portion. Blood could be seen in the glottis and he had had considerable hem-

optysis. Aside from the marked swelling there was not a single bruise or mark of violence externally over the neck. It was obvious that he had a fracture of the larynx, probably of his thyroid cartilage. The neck was not especially tender and he did not complain of pain, which is rather unusual in these cases. There were no external marks of violence other than the swelling.

A low tracheotomy was performed under local anesthesia. As it was impossible for him to breathe except with the head flexed forward, this position, together with the amount of air in the tissues, made the operation anything but easy. Multiple incisions were made in the skin to release the subcutaneous air over his thorax, shoulders, eyelids, etc. After ten days the laryngeal swelling had subsided and the local doctor discontinued the tube, allowing the tracheotomy wound to close. I did not see him again until six weeks afterwards. At this time he was perfectly all right except for some impairment of his voice. It has a peculiar vibratory quality and he complains that it is weak. Voice impairment is usual in these cases and is apt to be permanent. Examination of his larynx was essentially negative except for what appeared to be the healed scar of the fracture seen anteriorly just below the vocal cords.

Fracture of the larynx is always a serious injury, the prognosis, of course, varying with the extent of involvement of the cartilages. Tracheotomy would seem indicated to restore the airway patency and put the larynx at rest. In this case it was urgently demanded to avoid asphyxia.

Note: Since this has been written a very comprehensive paper on Fractures of the Larynx by Gardner has been published in the *Archives of Otolaryngology.*

Professional Building.

SYPHILITIC TRACHEOESOPHAGEAL FISTULA. REPORT OF A CASE.*

DR. ROBERT M. LUKENS and DR. JO ONO,
Philadelphia.

Tracheoesophageal fistula occurs with a certain frequency. However, syphilis as an etiologic factor is more or less unusual. Of 143 cases collected by Sirot[1] in 1898, 68 were due to neoplasm, 11 to foreign bodies, two to pressure of a tracheal cannula, four followed a cicatrix, 17 to inflammatory lesions, three to perforation of an ulcer and one each to aneurysm, sympathetic and trophic disturbances. In 36, the causes were undetermined.

In 1903, Connor[2] reported 128 instances of syphilis of the tracheobronchial tree, two of which, tracheal ulcers, perforated into the esophagus. Moersch's[3] series of 17 cases of acquired esophagorespiratory fistulae, observed in the Mayo Clinic during the period 1917 to 1930, contained no cases due to syphilis. We were able to find only 12 cases of tracheoesophageal fistula due to syphilis in the literature and there is a doubt about the origin of one of these cases.

The following case is one of syphilitic tracheoesophageal fistula of proven etiology.

Case Report: A negress, age 42 years, was admitted to the service of Dr. Louis H. Clerf at the Jefferson Hospital on Aug. 15, 1932, because of great difficulty in swallowing both liquid and solid food. This dysphagia developed suddenly ten days before admission and was associated with cough and expectoration, which included particles of previously ingested food. There was rapid loss of weight. There was found nothing in the family or personal history to suggest syphilis or foreign body.

Physical Examination: The patient was an emaciated adult negress. The face was thin and pinched; the eyes were sunken,

*From the Bronchoscopic Clinic, Jefferson Medical College Hospital.
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the lips parched and the skin dry and harsh. There was no dyspnea when the patient was quiet. The eyes were normal. There was oral sepsis and decayed teeth. There was no paralysis of the tongue or soft palate. The larynx was negative except for a slight pallor. The chest was long, flat and emaciated. Expansion was equal on both sides. Palpation and percussion elicited nothing abnormal. Moist rales were present at both bases. The abdomen was markedly distended, chiefly confined to the region of the stomach. There was no pain, tenderness, rigidity, nor signs of fluid. The reflexes were normal. The superficial lymph nodes, including the epitrachlear nodes, were palpable, small and hard. The temperature was 99° F., and the pulse 140 per minute. Respirations, 28 per minute on admission.

A special test devised by one of us (J. O.) was made to determine the nature of the disturbance in swallowing. The patient was placed successively in the recumbent position, on the right side, the left side and prone. While in each of these positions she was given a few sips of water to swallow. In the last three, the act of swallowing was accompanied by violent paroxysms of cough and expectoration of the swallowed fluid. This did not occur while lying in the recumbent position. A very brief period intervened between the swallowing of the water and the onset of the cough.

Roentgen examination was difficult because of the occurrence of severe paroxysms of coughing whenever the patient swallowed the barium mixture. For that reason films could not be secured. By fluoroscopy it was noted that the barium was displaced forward and entered the trachea, although a small quantity did pass into the stomach. The esophageal obstruction was near its upper extremity.

The Wassermann and Kahn tests of the blood were positive, plus four. The blood count showed hemoglobin, 65 per cent; erythrocytes, 4,000,000; leukocytes, 14,800. The sputum was repeatedly negative for tubercle bacilli.

A diagnostic esophagoscopy was performed and pus was traced down to an opening in the esophagus, which was partially covered by a necrotic flap (see Fig. 1). It was situated 21 cm. below the upper alveolar process, in the left anterior quadrant of the esophagus. Air was forced through this fis-

tula at each expiration and the necrotic flap blew in and out with the air current. The fistula was approximately two cm. in diameter. A Rehfuss tube for feeding purposes was introduced into the stomach through the esophagoscope.

The patient was given antileutic treatment; however, she became progressively worse. The temperature rose to 105° F., and symptoms of bronchopneumonia supervened. The patient died five days after admission.

Fig. 1. Schematic drawing illustrating the appearances found at esophagoscopy. The fistulous communication was observed on the left anterior quadrant of the esophagus. It was partially covered by a flap which moved with the respiratory blast, being forced upward into the mouth of the esophagoscope during expiration and cough.

Postmortem examination by Dr. C. J. Bucher showed that at the upper end of the esophagus there was an oval shaped perforation two cm. in its long diameter, which communicated with the trachea. The margin of the perforation was slightly elevated, firm, red and clean cut. The remainder of the esophagus presented no gross lesions. In addition there was found a gumma of the trachea and mediastinum, acute suppurative bronchitis and bronchopneumonia.

DISCUSSION.

The case presented illustrates certain features of tracheoesophageal fistula due to syphilis. Of these the extreme seri-

ousness of such a lesion is foremost. All of the reported cases, except two, terminated fatally, death being due in most cases to bronchopneumonia, putrid bronchitis or pulmonary gangrene.

An appreciation of the fact that syphilitic tracheitis and bronchitis are not altogether rare entities is of importance to the diagnostician. Direct examination of the tracheobronchial tree by bronchoscopic and by Roentgenologic methods and the employment of routine blood tests for syphilis in cases where there is evidence of a chronic tracheal lesion will go far in establishing the true nature of the disability if carried out early in the disease.

The esophageal symptoms and the palpable epitrochlear lymph nodes were the only manifestation of syphilis which were recognized clinically in our case. The syphilitic nature of the lesion was not determined, however, without the aid of the laboratory and of endoscopy. The differential diagnosis of syphilitic lesions of the trachea or esophagus are not always easy. Power[4] draws attention to the lack of symptoms in newly formed gummata of the trachea. There are, however, certain symptoms which should make the physician suspicious of such an ailment. If a patient has a history of syphilis or a strongly positive blood Wassermann reaction and increasing dyspnea over a period of time with paroxysmal cough and purulosanguineous expectoration, a luetic lesion of the trachea should be considered. On that evidence endoscopic examination of the trachea is indicated. The unfortunate accident of perforation of the esophagus is accompanied by a train of symptoms more or less constant. The perforation is sudden. It occurs frequently during ingestion of food and is usually ushered in by sudden choking and paroxysmal cough, apparently induced by swallowing. If in addition the patient coughs up food when swallowing, the symptoms are pathognomonic of the condition. The dysphagia accompanying a tracheoesophageal fistula is of a particular kind. Ordinarily, a patient with dysphagia has difficulty in getting the food down due apparently to some interference, mechanical or otherwise, with the normal act of swallowing. When fistula is present, the dysphagia is accompanied by violent choking. This paroxysm of choking is due to food or liquid passing into the tracheobronchial tree. There is a short but definite pause[7]

between the act of swallowing and the onset of the paroxysm of choking. It represents the period of time required by the food to reach the fistula. Another sign, dependent for its existence on the opening between the two structures, is the distension of the upper abdomen by air which passes from the trachea through the fistula and is swallowed with food.

Bronchoscopy and esophagoscopy, in conjunction with Roentgenology, are of the greatest value. In most instances the perforation can be distinctly viewed through the bronchoscope or esophagoscope or both. Other data can be obtained by such a procedure. Air will be forced out of the esophagoscope as long as its tip is proximal to the fistulous opening; after it passes the fistula, the expulsion of expired air ceases. This sign is more satisfactorily elicited with an endoscopic tube than with a rubber catheter as advised by T. Pantano.[5]

Beeler[6] was the first to employ Roentgenology in the determination of tracheoesophageal fistula. Since his report it has been used more often than endoscopy. By itself it often falls short of the desired result. As our case well illustrated, the swallowing of barium induced so much cough that satisfactory plates could not be obtained. The fluoroscopic examination, while indicated, does not always yield results conclusive as one should expect. A combination of endoscopy and Roentgenology should be employed in these cases, as either method by itself may fail in establishing a definite diagnosis.

Much valuable information can be obtained from clinical diagnostic methods. One of us (J. O.) reports a simple clinical sign which is present in tracheoesophageal fistula and can be elicited by any physician. This consists of cough and expectoration of swallowed material shortly after the act of swallowing while the patient is sitting upright or lying on the right or left side or prone, but no cough nor expectoration when the patient is lying on the back. Tracheoesophageal fistulae are always on the anterior esophageal wall because that is the party wall between the trachea and esophagus. Hence fluid, when swallowed, will not pass readily through the fistula into the trachea when the patient is lying on the back, but will when the patient is in any of the other four positions.

SUMMARY.

1. Tracheoesophageal fistula of syphilitic origin is uncommon.

2. The importance of endoscopic and Roentgenologic examination is discussed.

3. A new clinical sign, which is elicited in tracheoesophageal fistula, is described.

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1923 Spruce Street.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of December 20, 1933.

(Continued from a previous issue.)

The Regression Theory of Otosclerosis. Dr. L. K. Guggenheim.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. EDMUND P. FOWLER: Mr. Chairman, ladies and gentlemen: I am sorry that I cannot speak the language of Dr. Guggenheim. I am afraid that my discussion will seem very ordinary, but at least you can understand all my words.

Dr. Guggenheim is a hypothesist who sets forth clearly the reasoning and facts upon which his hypothesis is based. I am sure he welcomes criticism from every angle and we would not be at all surprised to see him at any moment set up another hypothesis as to the causation of otosclerosis, as we all have done.

Anatomical studies alone will never solve the problems of medicine, but linked with logical suppositions successfully proved or disproved, such studies will eventually lead somewhere near the truth.

It is always stimulating to ramble through the realms of gene propelled dystrophies when other paths seem blocked, but even granting the presence of regressive tendencies does not get us anywhere, in so far as management or treatment is concerned. Nature does not produce perfect offspring even though the parents be perfect, and I do not believe in birth control or abortion to control otosclerosis. In the long run we have got to take the bad with the good. Nature's laws will prevail.

Bilateral and even symmetrical involvement is also very frequently seen in middle ear inflammation. The reason that bilateral involvement is not more frequently diagnosed is because we are often hurried or careless in our examinations. We do not look. Now it may be that inflammation also can be a gene propelled affair, but in any event, gene propelled or not, there must be also a determining cause, not only for the beginning, but also for the continuance, arrestation, or recurrence of the lesion growth. In other words, we must hunt for "the determining cause," which must at times vary markedly even in brothers and sisters apparently in the same environment. Otherwise, how account for the variations in incidence of otosclerosis in these?

It is difficult to conceive of a regression when so often lamellar bone is present in otosclerotic foci. Lamellar bone is more highly developed bone than the normal endochondral capsule in which the otosclerosis develops.

Otosclerotic-like bone has been produced by venous stasis. Chronic passive congestion in any tissue causes some inflammatory reaction, and the areas of predilection for otosclerosis are no exception. The statement that leukocytes are absent in otosclerosis is not wholly accurate, because we find phagocytic or large wandering cells, histocytes, as well as foreign body giant cells (osteoclasts). All these cells really represent types of leukocytes.

And so it comes down to what we really mean by inflammation and irritation and so forth. As reported we have found evidences of inflammation in every case of otosclerosis that we have examined under the microscope (fifty odd cases). The slides of fourteen of these cases were obtained abroad and thirty-four from the collection of Johns Hopkins Hospital. Our clinical evidence in 128 carefully studied cases shows signs of past inflammatory lesions in the middle ear in 75 per cent, and that there were no blood abnormalities of any kind which could be linked up with the otosclerotic process. In other words, the pathological evidence substantiated the clinical observations. With the evidence of universal inflammatory reactions in the ear, it would seem folly to neglect these as at least contributing factors in the etiology of otosclerosis.

Sometimes the thing for which we are looking is so close at hand that we are apt to look right past it and wander into distant fields. I am tending more and more to the opinion that otosclerosis cannot be separated entirely from the inflammatory reactions.

DR. LOUIS K. GUGGENHEIM: I have already taken too much of your time. I enjoyed the discussion by both Dr. Duel and Dr. Fowler very much. I think for the present at least, that Dr. Duel is quite correct, that the only way in which otosclerosis can ever be eradicated or controlled will be by controlling marriage and pregnancy; until someone finds a way to influence the process of ossification in the developing fetus by treatment of the mother, and that is a long way off.

Dr. Duel mentioned that other bones may be involved. I do not know of anyone who has really systematically examined the other bones in cases of otosclerosis. It is a dream plan we have all had because we really do not know whether other bones are involved or not. We like to say they are not; that this process is confined to the ear mechanism. We may find some day that Weber is right, that it is part of a generalized involvement of the osseous system.

Dr. Fowler I have known for a long time leans toward the inflammatory theory. It is difficult to understand how the involvement at the internal auditory meatus can result from an inflammatory condition, unless we look upon that as a result of a petrositis in the embryo. As far as evidence of inflammation is concerned, I have never seen anything in the way of inflammation, such as round cell or leukocytic infiltration. Possibly Dr. Fowler is correct in speaking of the histocytes and giant cells as being evidence of inflammation. I have looked upon the giant cells as a fusion of the osteogenic cells, but I do not know. I think you will all have gleaned from this paper and the discussion that there remains much work to be done.

DR. SAMUEL J. KOPETZKY: Here, for the time being, we will leave otosclerosis, to resume it again in future studies.

Our next speaker is a man who has come a long distance, a distinguished practitioner in otolaryngology and a student. He is going to present the subject of osteomyelitis of the skull. I take great pleasure in introducing to those who do not know him, and welcoming with those who do, Dr. A. C. Furstenberg, Professor of Otolaryngology at the University of Michigan Medical School, Ann Arbor.

The Pathology of the Spread of Osteomyelitis of the Skull. Dr. A. C. Furstenberg.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. IRA COHEN: Mr. Chairman and members of the Section: May I first express my appreciation for the opportunity of discussing this very instructive and very clear-cut paper by Dr. Furstenberg. My interest in osteomyelitis

of the skull arose, so to speak, from inside of the skull rather than from the outside, and I leave any remarks directed toward the frontal sinus to those gentlemen who follow me and who know a great deal more about it than I do. In fact, only about a little over one-half, I would say, of the 16 cases I have observed in the past four or five years have been of frontal sinus origin. Some have been traumatic and some have been metastatic. The metastatic group tends to act very much the same as does the osteomyelitis, starting from the frontal sinus. The traumatic group have a greater tendency to limit their spread.

With Dr. Furstenberg I am in full accord that the spread of the disease from the sinus takes place by more than one channel. Of course, we may have a spread by continuity. To my way of looking at it the greatest spread is through the diploic veins. Unless one accepts absolutely the dural spread, it is difficult to understand how one often sees scattered foci, six, seven or more centimeters apart, from the frontal to the occipital region with normal bone in between. I have often asked myself why, with this marked invasion and thrombosis of the diploic veins, one does not more often see in osteomyelitis of the skull thrombosis of the larger venous sinuses. Of course, it has been described, but why doesn't one see it more often, or why doesn't one have a general bacteremia in the face of this general infection with the general picture at least of multiple abscesses?

The spread along the dura is seen rather frequently, and yet I have made rather conflicting observations, so to speak; as where there is an osteomyelitis seemingly involving only the outer table, and as is my wont I removed both the outer and inner tables and found perfectly normal dura underneath, even though that focus is as far removed as the frontal eminence is from the frontal sinus. I have seen granulations in the dura starting from the point of obvious bone disease and extending forward four or five centimeters to a true epidural abscess, with the bone overlying this granulation tissue perfectly normal.

I was particularly interested in Dr. Furstenberg's remarks about bone regeneration, and especially his statement that perhaps it is a mild infection which prompts this regeneration. In our cranial surgery it is rather the exception than the rule to find bone regeneration. We remove, as you know, for subtemporal decompressions sometimes rather wide areas of bone. Occasionally we have to remove the entire flap, not often, but occasionally, when there is very marked pressure. I recall one instance in a woman of twenty-odd years—I particularly mention the age—in whom after removal of the tumor I had to remove the flap, and in whom I thought I would attempt a careful suture of the edges of the periosteum in addition to the usual galea sutures and skin. It was studied at a subsequent operation a year later. Two years since this subsequent operation there has been absolutely no bone regeneration. I think from the standpoint of cranial surgery we see the formation of new bone most often in the cases where bone has been removed for depressed fractures. I had not thought of it from that standpoint, but it is possible that in those cases where it does occur it may well be that there has been a compound fracture with a mild infection.

From the standpoint of treatment I think Dr. Furstenberg is a little more radical than I want to be, although I must say I do certainly advocate the removal of both outer and inner tables, and the removal of obviously diseased bone. However, I have made no effort to go very wide of the disease, and my reason for that is that not infrequently I have seen a focus appear, following a frontal sinus infection, say over the frontal eminence, and remain limited to an area the size of a ten cent piece. That has been the only focus of osteomyelitis, with no further disease of the bone. Thus of course I am unable to prognosticate as to which case is going to spread and which is going to remain localized. For that reason I prefer to remove only the part which is diseased.

I would like to ask Dr. Furstenberg one question. Does he believe that this newly formed bone has diploic channels?

I think it is very obvious from the paper we have heard this evening that osteomyelitis of the skull is a dreadful disease and much to be feared. It is only by such reports and studies as Dr. Furstenberg has made that we are going to see our way as to how to treat it properly.

DR. ROBERT E. BUCKLEY: Mr. Chairman and gentlemen: I was amazed, delightfully amazed, at the remarkable presentation Dr. Furstenberg gave us tonight. Certainly the Section owes a debt of gratitude because the doctor has taken up with us this complication of sinusitis which has become practically synonymous with death. As recently as 1921 one of the English writers, Maclay, mentioned that in a review of the literature the postoperative osteomyelitis of the skull that he had been able to collect had shown a mortality of 100 per cent. They differentiated then, and I think many do still, between postoperative and preoperative osteomyelitis. The postoperative had by far the greater mortality and was to be more feared. Another reason Dr. Furstenberg is to be congratulated is that since the disease is so insidious, and takes so long before its termination, the natural tendency is to be more conservative. Radicalism demands a type of bravery very few of us possess. For that, too, he should be congratulated. The entire topic comes down to what the last speaker, Dr. Cohen, mentioned, the question of radicalism against conservatism. I have had no experience in radicalism, but I have had, unfortunately, a certain amount with conservatism.

I just want again to congratulate Dr. Furstenberg on his results.

DR. RUDOLPH KRAMER: Mr. Chairman, members of the Section, and guests: I am personally very happy to be here and to hear Dr. Furstenberg's paper, because so many of the viewpoints he has and so many of his experiences totally coincide with my own, based upon a series of cases of which approximately 12 cases were diffuse osteomyelitis of the skull.

I think the discussion is not on the perforations of the various walls of the sinuses with more or less necrosis of the walls, but on the diffuse spread over the calvarium. I have always felt that the main propagation of the lesion is a vascular one. In the past two years I have had what I think is some confirmation of this. In two cases, within twelve hours of the onset of symptoms of the sinusitis—mind you, not of the osteomyelitis, but of the sinusitis—the patient had fever and severe supraorbital headache. There was a localized edema over the frontal eminence which subsided within 24 hours. Both of these cases were operated on some time later for an osteomyelitis, and in both it was found at the site of the early swelling over the frontal eminence that there was a perforation, due to the osteomyelitis, through the external table with an extension of the disease further on. Furthermore, at operation in these cases practically every one of them shows a spread from the angle between the anterior and posterior walls, either at the upper limit of the frontal sinus or at the lateral or mesial angles. I have always regarded this as the start of the thrombophlebitis and that from there on the spread of the disease over the calvarium occurred. I think the point which Dr. Furstenberg brought out, about the diploic veins supplying the inner table and being important in propagating or advancing the lesion in the inner table, is important in causing the lesions which so often occur in these cases.

From my experience, all the cases of osteomyelitis of the skull, with the exception of two I have seen, have been spontaneous in origin. By that I mean that they have not followed any operation or intranasal manipulation. I think it is a peculiar experience, because in the literature most of the men have found that it has followed an operation of some sort. It may be we have waited longer before operating. It may be we do not operate on frontal sinusitis unless there are complications, such as a perforation or a question of an intracranial lesion. At any rate, with the exception of two cases, they all followed sinusitis unoperated and untouched.

As I said before, from these two standpoints, I believe that the osteomyelitis starts almost spontaneously with the onset of the sinusitis, that there is a phlebitis of the vessels of the sinus which spreads over the calvarium. I think we have a very good analogy in the osteomyelitis of the sphenoid bone. In ten cases which I described here several years ago, all of them unoperated cases with the exception of one, in which the diagnosis of osteomyelitis had been made before the operation was performed, most of them had an osteomyelitis of the basi occiput or basi sphenoid. There again we have the spontaneous origin of the osteomyelitis.

As far as the treatment is concerned, I am in entire accord with Dr. Furstenberg. I know that many cases have been allowed to wait until they presented an external abscess which has then been drained and perhaps the external table has been taken off, and the patient has gotten well. However, I think by and large that the only safe procedure is to remove the entire thickness of the bone, because after all patients die from intracranial complications as a rule. The usual cause of death is either meningitis or a brain abscess. There is no way of determining the presence of these complications and no way of preventing their formation except by removing both tables. For that reason I think that in addition to going as widely as possible, both layers of bone should be removed. Very often intracranial complications are totally unsuspected and are only apparent when the dura is uncovered. As far as preventing osteomyelitis following operation, I do not think we have any method now except one. Be as gentle as possible and destroy as little tissue as possible, particularly as one gets near the upper limit of the frontal sinus.

DR. HARMON SMITH: Mr. Chairman and gentlemen: I should like to take this occasion to express in behalf of the Section, our appreciation of the two papers presented to us tonight by Dr. Guggenheim and Dr. Furstenberg, and to assure them that although we are delighted to listen to their papers and to agree with them up to a certain point, that we claim, however, the privilege to disagree with them on some points without incurring any animus which is a recognized scientific privilege. Dr. Fowler does not entirely agree with Dr. Guggenheim. He merely means that in science there is no friendship nor hospitality that would bend his expressions from what he believes. We want to get at the facts and we want to deduct from what is presented the abstract truths of material, irrespective of what anyone may express or from wherever they may come. In my remarks, written before coming here, I am going to differ with the presenter of the paper on osteomyelitis, and I am justified in so doing for about the same reason as the man who wrote that beautiful poem, "Home, Sweet Home"—because he had no home. I have had only three cases of osteomyelitis of the skull and they did not involve the brain. I have seen in addition, however, several at the Manhattan Eye, Ear and Throat Hospital, most of them in my own clinic and others in other clinics.

I have studied up the subject since Dr. Kopetzky asked me to discuss this paper and have inquired from others about it, so that some of the things embodied in this abbreviated paper represent some of the features of osteomyelitis that the doctor has already presented, but were unknown to me before hearing his paper; and one feature is particularly advocated as a possibility which it appears he has already employed.

After this composite and scientific presentation of the subject, there is little left for me but to emphasize the outstanding and conspicuous factors appertaining to it.

The early writers from Percival Pott, who first described this condition in 1760 and from which we derived the name of Pott's Puffy Tumors, to within the laft few decades, have all felt that either syphilis, tuberculosis, or some constitutional dyscrasia was an underlying factor in such conditions; but in 1913, Dan MacKenzie published a paper enumerating forty odd cases in which syphilis seemed to play no decided part. Later writers, as Skillern, Kernan and others have almost definitely determined for us that neither syphilis, tuberculosis nor operative interference are in any definite or abstract measure responsible for osteomyelitis. Among these writers we should by no means omit

Dr. Joseph Bryan of Washington, who has been a keen observer along this line, who has reported several cases and who has had a number of cases which he has not yet reported—one of which was a Mr. Prosser, a brother-in-law of our late lamented fellow, Dr. Fetteroff, in which instance an X-ray was made two hours before operation and which disclosed an extensive osteomyelitis of the frontal bone, thus showing that operative measures were not an etiological factor here.

Therefore, we can conclude with reasonable certainty that osteomyelitis is due to an infection of the bone, the predominating organism being the streptococcus hemolyticus. It is also a thrombophlebitis, and the infection therefrom is carried into the diploe of the skull and finally results in either a localized or a general necrosis. The skull in this locality lends itself peculiarly to the spread of invasion through the Haversian canals.

Knowing the pathology of the disease, knowing the character of the bone that lends itself to its wide distribution, knowing also that it extends to more remote areas in adults than in young persons, what might be available surgically to control the same? In conflagrations in cities a widely dynamited area ahead of the fire usually keeps the fire within controlled limits. In forest fires, a fire break or the destruction of forest of sufficient width often prevents further extension. So here, when we have a conflagration of infection with Haversian avenues facilitating its extension, there is but one method available to circumscribe its extension. The coronal sutures in the adult are too intimately enmeshed to offer material obstruction to the advance of diploic infection. Hence I would offer as a suggestion, though to my knowledge not as yet applied, a channel, ditch or canal chiseled through the frontal bone parallel to the coronal sutures and materially removed from the objective focus of involvement. This channel should extend through the skull to the dura mater. Likewise I would suggest that the proximal wall of this ditch be completely effaced by bone wax, so that infections traveling by the Haversian routes would meet obstruction of their exits toward the distal bony wall. Mere trephining will not prohibit this infection from passing between the shell holes of the trepanned area, but the fire line of the ditch would prevent further encroachment upon healthy bone.

In pursuing this subject in textbook literature, I am amazed at the dearth of expression appertaining thereto. There appear years of dehiscence when either osteomyelitis of the skull did not occur, or when no one commented thereon. Bosworth does not mention it in his work on nose and throat. Kyle gives one line of about eight words thereto. Ingals, Phillips, Ballinger, Wright and Smith have nothing to say upon it. Should we deduct from these writers and their paucity of expression that no such thing existed and conclude therefrom that later operative interference of more radical nature played some part in the etiology of the disease? I am not of this opinion, however much I am aware of a greater number of these cases now than when I first began the practice of rhinology.

In the last four or five years there have been even a greater number of these cases occurring in the clinics, and I am led to believe that economic conditions have imposed upon the clinical patient a necessity for less immediate attention to his condition than before. The patient appears now with a sinusitis further advanced and even with a possible osteomyelitis, before operative measures have been suggested. Under such conditions I would advocate an extensive uplifting of the scalp and application of surgical intervention by chiseling a bony ditch materially in advance of any objective involvement of the bony structure.

I should like to ask several questions. First, why should not osteomyelitis follow mastoid disease, particularly as the tables of the mastoid cavity have been exposed to infection longer than the sinus walls? Secondly, why should not osteomyelitis result from long continued neglect of antral involvement?

In the frontal bone there seems to be a peculiar condition of the bone which aids and abets this peculiar tendency to infection, and this appears to be due

to the diploic character thereof and the Haversian canals. I cannot see that the dura has any more to do with it than it would in the mastoid cavity.

DR. A. C. FURSTENBERG: Mr. Chairman: I am very grateful for the excellent discussion of this subject. I wish to say in answer to Dr. Cohen's question concerning the histological structure of the new formed bone that it presents diploe after regeneration is complete.

Dr. Smith has pointed out the value of the furrow cut in the bone posterior to the infection, and I have no doubt that this technique would have a distinct value were the furrow made widely enough. In the case I described, the surgeon had cut a furrow in the frontal bone four months before the infection spread to a point where it caused death. At the time of the operation bone dust from the furrow showed no evidence of osteomyelitis. Moreover, the margin of healthy bone had been sealed with bone wax. At postmortem a piece of bone taken from the distal margin of the furrow revealed an osteomyelitis. In some manner the infection had spread across the furrow, causing extensive involvement of the cranial vault. I strongly suspect from a number of pathological observations I have made that the spread of infection was on the surface of the dura through the efferent diploic veins.

Dr. Smith has asked a very good question. Why does not osteomyelitis start from mastoid disease? It is probably very rare. I have a slide of a very amazing case in which it did start from mastoid disease and spread over a large part of the calvarium. We removed a large portion of the skull and there has been restoration of the defect almost to normal. In the literature there are very few reports of such etiology, although I am convinced that in this particular patient the infection started from an acute exacerbation of a chronic mastoiditis.

DR. HARMON SMITH: How about the long bones, as for instance osteomyelitis of the tibia, which never gets well permanently but only temporarily?

DR. FURSTENBERG: I do not know. I cannot answer this question.

DR. LOUIS K. GUGGENHEIM: I would like to know Dr. Furstenberg's explanation of why we do not see osteomyelitis of the skull more often following mastoid disease. I would also like very much to see the slides of which he spoke.

DR. FURSTENBERG: I purposely tried to avoid this question. I frankly admit that I do not know. I have studied it from a number of angles, but thus far I have been unable to determine why it does not start more frequently from mastoid disease. I will present a case which was under my observation from the beginning in which the osteomyelitis started from an infection in the mastoid bone.

Lantern slides of a case of osteomyelitis resulting from an acute exacerbation of a chronic mastoiditis were demonstrated. Here the infection started in the temporal bone, then spread to the occipital bone and subsequently involved a large part of the skull. The osteomyelitic lesion was extensively removed, followed by rapid regeneration of bone until the entire cranial defect was almost completely closed.

DR. SAMUEL J. KOPETZKY: Just to complete the record and answer the last question, it may be that osteomyelitis of the skull does not take place from mastoid infection, because in a majority of mastoid infections you are dealing with nothing more than an osteitis. In those rare types of ear infection in which you have bone marrow and Haversian canals, that is, you have a diploic process, not a pneumatic process, then there is present the basic structure upon which the osteomyelitis can develop. That may be one of the factors that accounts for the rarity of its occurrence in the average garden variety of ear infections.

I want to express to the gentleman from St. Louis and the gentleman from Ann Arbor my profoundest gratitude to them for their masterly presentations this evening. They have provoked us to further thought and further work, and for that, even more than for their masterly presentations, we are indebted to them.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of Jan. 15, 1934.

Dr. F. E. Hasty, Chairman, Presided.

An Atypical Case of Peritonsillar Abscess. Dr. Guy M. Maness.

R. Mc., a white male, age 13 years, was first seen on Jan. 2, 1934. He was brought for examination by his aunt, who, because of a speech defect which had developed in the past month, had looked in his throat and discovered a large mass on the right side. The boy had not complained of pain or sore throat. His family did not think he had been having any fever. They complained that they could not keep him in the house. He was out playing with other children every chance he had. The speech defect had been present for the past month. He had regurgitation of fluids through his nose on swallowing. He had no difficulty in opening his mouth and there had been no salivation. The speech defect and difficulty of swallowing had been progressive. The patient did not remember having had a definite sore throat at the onset of the trouble; however, he had the history of frequent sore throats.

The examination revealed marked increase in the size of the right tonsil, it being pushed beyond the median line. There was no definite hyperemia of the tonsil, its pillars or the pharynx. No fluctuation was detected on palpation of the mass. The anterior pillar and soft palate showed no evidence of edema. In fact, it gave one the impression that the mass was due to increase in tissue rather than edema or accumulation of pus. The mass was very firm and was not definitely tender or painful on palpation. There were enlarged, firm, non-tender glands at the angle of the mandible on the right. The glands were moderately enlarged in the left side of the neck. The left tonsil had a fairly normal appearance. The nose, nasopharynx and ears were negative. The general physical examination was negative except for malnutrition and anemia.

Laboratory examinations: Urine negative except for 1+ albumen, red blood cells 3,840,000, white blood cells 16,000, hemoglobin 12 grams. Differential count was: P.M.N. 73 per cent, P.M.E. 2 per cent, P.M.B. 1 per cent, lymphocytes 19 per cent, large monos 5 per cent. The red blood cells were normal in size, shape and staining qualities. The platelets were normal. Wassermann was negative, stool examination negative, tuberculin test negative. Chest plate was normal. The temperature was 100°.

A diagnosis of probable lymphosarcoma of the tonsil was made when the patient was first seen. In the presence of the leukocytosis and some fever the possibility of a chronic peritonsillar abscess was also considered, but the former was thought to be the most likely.

It was decided to take a biopsy or to drain the abscess if one was present. 1 per cent novocain was injected in the anterior pillar on the right side. The needle was then inserted deeper into the peritonsillar tissues and about three cc. of novocain injected. This was followed with severe pain in the right tonsillar area, side of face and ear The anterior pillar was then incised in the usual area where a peritonsillar abscess is drained. Immediately a flow of thick white pus occurred. The incision was enlarged with a hemostat and about one ounce of pus was evacuated. There was rapid regression of the size of the right tonsillar area with improvement in swallowing and the return of normal enunciation. The wound healed rapidly. The culture of the pus showed staphylococcus albus. The biopsy showed only an inflammatory reaction.

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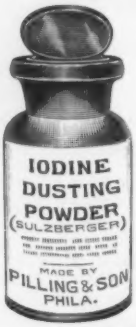

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